Clinical Electrocardiography

WILLIUS

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BY

FREDRICK A. WILLIUS

B. S., M. D., M. S. IN MEDICINE

Section on Clinical Electrocardiography
The Mayo Clinic, Rochester, Minnesota
and
The Mayo Foundation, University of Minnesota

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PREFACE

THE constantly increasing use of the electrocardiograph among clinicians, especially in hospitals and clinics, prompted the writing of this book. Much of the recent knowledge pertaining to cardiology has been obtained by careful study of the cardiac mechanism, normal and abnormal, as revealed by graphic methods of precision. The diagnosis of arhythmias, the localization of conduction lesions, and the identification of obscure tachycardias have been greatly simplified by electrocardiographic study. Much valuable knowledge pertaining to prognosis has been obtained, particularly with reference to abnormalities of the Q R S complex and to negativity of the ventricular T wave in isolated and combined derivations. The attempt must always be made to translate the facts that are determined graphically into practical clinical knowledge. Electrocardiography must always remain subsidiary to practical methods of physical diagnosis, but its value as a most important clinical adjunct cannot be too strongly emphasized.

I have attempted to present the subject of electrocardiography in a logical manner and to consider the fundamental principles, the technic of obtaining records, disorders of the cardiac mechanism, organic and functional, and, when data were available, facts regarding prognosis.

An effort has been made to obviate the difficulties confronting beginners by correlating physiologic and pathologic aspects of cardiac disease and by simplifying the classification of disorders of mechanism.

The bibliography is not complete, but the references chosen largely are of distinct contributary value.

FREDRICK A. WILLIUS.

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CHAPTER I

PHYSIOLOGIC CONSIDERATIONS

THE muscle of the heart differs anatomically and physiologically from skeletal muscle. The fibers are striated, interspersed with

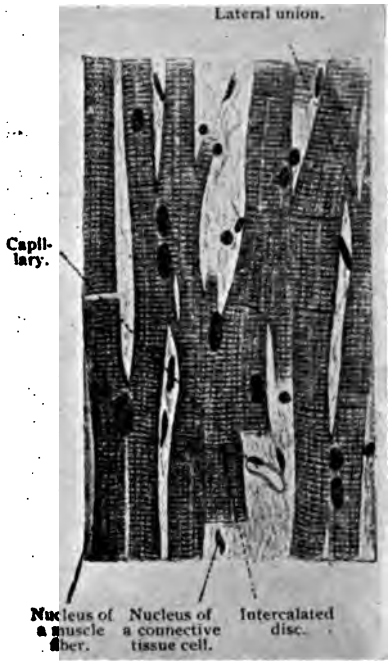

Fig. 1.—A longitudinal section of a papillary muscle of the human heart. × 360. (After Stöhr.)

very little connective tissue, and their bundles freely anastomose (Fig. 1). Three physiologic characteristics distinguish heart

muscle from other muscle: (1) heart muscle is constantly undergoing rhythmic contraction; (2) the response of heart muscle to stimuli is not proportionate but always maximal, constituting the "all or none" law of Bowditch, and (3) heart muscle in the state of contraction is insensitive to further stimulation, and is said to be in the refractory stage (Marey).

FUNDAMENTAL PROPERTIES OF HEART MUSCLE

The fundamental properties of heart muscle are: (1) rhythmicity, the power of impulse production; (2) irritability, the power

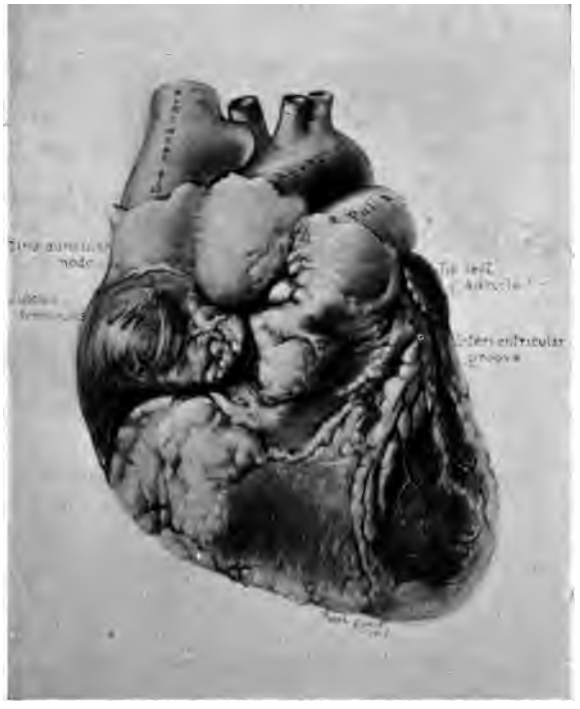

Fig. 2.—Heart, anterior view, slightly from right.

to respond to stimuli; (3) conductivity, the power of impulse transmission; (4) contractility, the power of adapting its shape

and length in response to stimuli, and (5) tonicity, the power of sustained partial contraction in which stretching is resisted.

Origin and Course of the Cardiac Impulse.—The sino-auricular node, a collection of specialized tissue (primordial tube remnant), lies in the sulcus terminalis at the juncture of the superior vena cava and the right auricular appendage (Fig. 2). It is composed of delicate, spindle-shaped, interlacing muscle-fibers and a few ganglion cells in a connective-tissue reticulum. The sino-auricular

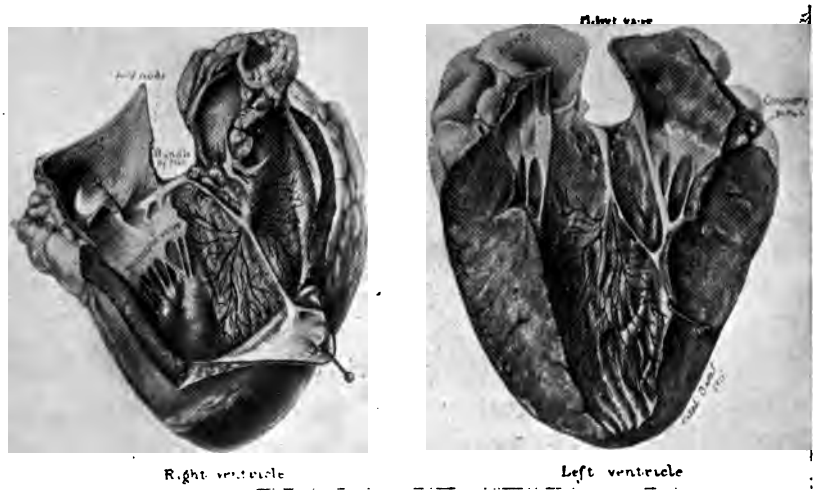

Fig. 3.—Right ventricle and left ventricle. Ventricular conduction system represented schematically.

node is the seat of origin of the cardiac impulse, as has been demonstrated by the investigations of Keith and Flack, Wybauw, Lewis, Oppenheimer and Oppenheimer, Brandenburg and Hoffmann, and Ganter and Zahn. It is the region of primary cardiac electronegativity. The excitation wave spreads from the sino-auricular node to the auricles and then reaches the auriculoventricular node.

The auriculoventricular node (Tawara) is situated in the right lateral aspect of the auricular septum, just posterior to the septal cusp of the tricuspid valve (Fig. 3). From the anterior extremity

of this node the auriculoventricular bundle (His) takes its origin, passes downward and backward, and at the level of the membranous septum divides into a right and a left branch. Each branch spreads in a fan-like manner in the subendocardial tissue of the ventricles, divides, subdivides, and intercommunicates, forming the subendocardial arborization system or Purkinje plexus. These fibers ultimately coalesce with the individual muscle-fibers of the ventricles. Thus the cardiac impulse has a path of conduction from its origin in the sino-auricular node to its destination in the ventricular musculature.

Origin of the Heart-beat.—Progress along physiologic lines is determining more and more the relationship of vital phenomena to biochemic reactions. The isolation of the thyroid active principle, thyroxin, by Kendall, and the quantitative physiologic reactions involved in its action are a typical example.

With this conception in mind it is logical to believe that the rhythmic cardiac impulse results from a biochemic reaction. Bredig and Wilke have observed rhythmic chemical reactions in the catalysis of hydrogen peroxid by metallic mercury. The action of hydrogen peroxid on mercury produces a yellow or brownish film of an oxid of mercury. The film is present for an instant, becomes dissolved, and leaves the surface of the mercury bright; the reaction is repeated. The liberation of the hydrogen bubbles was recorded by the use of a tambour and tracings were obtained resembling those of the heart.

MYOGENIC AND NEUROGENIC THEORIES

Whether the vital reaction which originates the cardiac impulse acts directly on muscle (myogenic) tissue or on nerve (neurogenic) tissue has been the subject of extensive controversy. The intimate association of nerve and muscle tissue incorporated in the myocardium makes it possible to give credence to both theories.

MUSCULAR ARRANGEMENT OF THE VENTRICLES

The arrangement of the muscle-fibers of the ventricles has been shown by Mall to be intricate. The inner fibers comprise the papillary system, which includes the right and the left anterior and posterior papillary muscles and the internal longitudinal fibers of the ventricles. The papillary muscles are thick bundles, the anterior being the larger, and they give rise to the chordæ tendinæ which insert into the auriculoventricular valve leaflets.

The outer or circular musculature consists of two distinct spirals, one running from the tricuspid region of the heart to the apex of the right ventricle (sinospiral), and the other from the aortic and mitral ring to the apex of the left ventricle (bulbospiral). Each spiral is composed of a deep and a superficial layer, opposed at right angles to one another, and communicating by strands running from the papillary muscles of one ventricle to those of the other. The work of Hering indicates that the papillary muscles are the first ventricular structures to enter into contraction.

CARDIODYNAMICS

Just before systole of the ventricles the auriculoventricular valves are open, while the semilunar valves are closed. With contraction of the ventricles the pressure in these chambers is raised and the auriculoventricular valves close abruptly. There is thus a short period (0.07 to 0.09 second) early in systole when all four valves are closed. During this period blood flow in the auricles and ventricles ceases. This is termed the "presphygmic period" and terminates when intraventricular pressure rises above the pressure in the arterial circulation. The outflow of the ventricles during systole, according to Henderson, is quite continuous and occupies about nine-tenths of the period. Filling of the chambers occurs during diastole, and the length of diastole is determined by the rate of contraction.

The diastolic period is divided into two parts: (1) diastole proper, during which the filling of the ventricles is effected, and (2) diastasis, during which little or no filling occurs. Hirschfelder emphasizes the fact that "the greatest amount of output in unit time occurs at a rate which just allows the phase of diastolic filling to be complete, but in which the next beat occurs before diastasis sets in. Any rate above or below this brings about some slowing of the circulation."

THE CARDIAC NERVES

The heart is under the control of two sets of nerves (extrinsic innervation), the vagi connecting it with the medulla and the accelerator nerves connecting it with the sympathetic system. These, in turn, divide into branches communicating with the cardiac ganglia (intrinsic innervation, Fig. 4). Each vagus and each accelerator is distributed largely over the corresponding half of the heart, although free anastomosis between the halves occurs.

The Vagi.—These arise from the motor and sensory nuclei in the medulla. Two sets of fibers are contained in the vagi, the afferent or sensory fibers, which carry impulses from the heart, and the efferent or motor fibers, which convey impulses to the heart.

Afferent Impulses.—It has been demonstrated that a relatively slow wave of electronegativity passes upward with each heartbeat. In this manner impulses reach the higher centers to be converted into subjective sensations.

Efferent Impulses.—The efferent impulses which pass down the vagi affecting the heart are: (1) the inhibitory influence slowing rate (negative chronotropy); (2) the influence diminishing strength of contraction (negative inotropy); (3) the influence diminishing irritability (negative bathmotropy); and (4) the influence diminishing conductivity (negative dromotropy).

The Accelerators.—The accelerator nerves are derived from rami communicantes from all the cervical and upper four thoracic nerves which pass to the superior, middle, inferior, and stellate ganglia. The accelerator function has been studied electrocardiographically by Rothberger and Winterberg, who conclude: (1) the accelerator action of the right nerve is greatest because of its

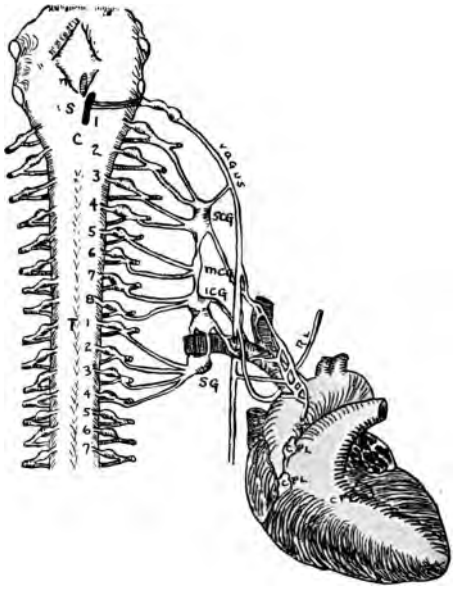

Fig. 4.—Origin and distribution of the cardiac nerves: *M* and *S*, Nuclei of the efferent (motor) and afferent (sensory) fibers of the vagus; *C*, 1, 2, 3, 4, 5, 6, 7, 8 and *T*, 1, 2, 3, 4, 5, 6, 7, cervical and thoracic spinal nerves; *SCG*, *MCG*, *ICG*, superior, middle, and inferior cervical ganglia; *RL*, recurrent laryngeal nerve; *CPL*, cardiac plexus. (Modified from Hirschfelder.)

distribution to the sinus region; (2) stimulation of the various branches of the right cervical sympathetic gives rise to an electrocardiogram showing overaction of the right ventricle, while stimulation of the left cervical sympathetic fibers gives evidence of an overacting left ventricle; and (3) stimulation of certain cardiac branches of the left cervical sympathetic gives rise to coincident contractions

of auricles and ventricles by causing the impulse to arise in the auriculoventricular junctional tissues.

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CHAPTER II

ELECTROCARDIOGRAPHY

ELECTROCARDIOGRAPHY is dependent on the physiologic law that when any muscle is stimulated, the portion to which the stimulus is applied goes into contraction, and this area becomes electronegative to the resting portion. As early as 1855 Kölliker and Müller demonstrated by means of the capillary electrometer

Fig. 5.—Cambridge model electrocardiograph: *A*, Camera mechanism; *C*, lantern; *D*, electromagnets, microscopes, and fiber case; *E*, power control board; *K*, time marker; *L*, water-cooled condenser.

that electric currents are produced with each beat of the heart. The introduction of the string galvanometer by Ader, in 1897, opened the portals to electrocardiography.

The galvanometer is based on the physical law that a current produces a magnetic field which acts at right angles to its course, varies with the intensity of the current, and attracts or repels

proportionately another neighboring magnetic field. The electrocardiograph is but a modern elaboration of the original string galvanometer (Figs. 5, 6).

The fiber of the electrocardiograph is a delicate quartz filament from 0.003 to 0.005 mm. thick, it is barely perceptible to the naked eye, and is coated with gold or silver to permit con-

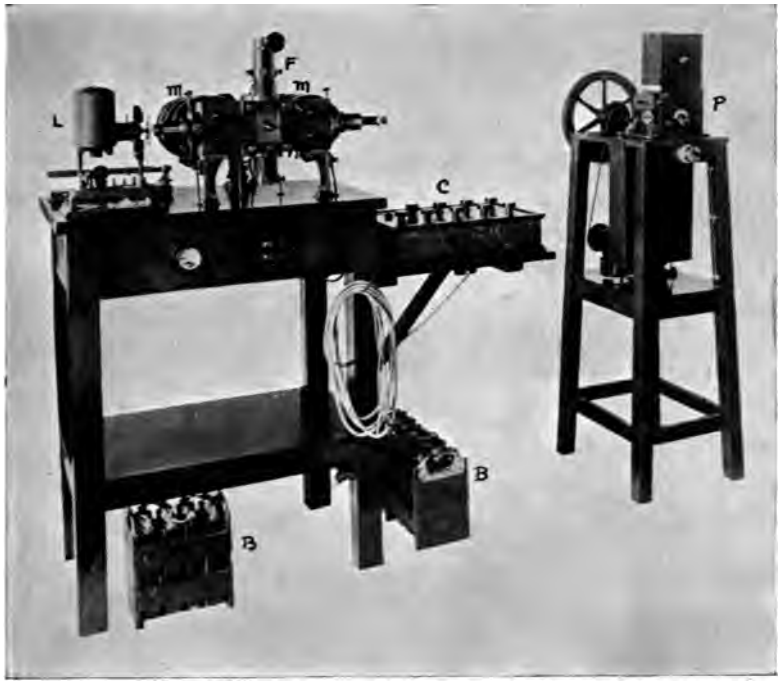

Fig. 6.—Hindle model electrocardiograph: *B*, Batteries; *C*, control board; *F*, fiber house; *L*, lantern; *M*, electromagnet; *P*, camera.

ductivity. It is suspended between the poles of a powerful electromagnet (Fig. 7). The movements of the fiber, activated by cardiac action, are recorded by projecting its shadow, magnified by a high-power microscope, on a camera mechanism. The time is recorded by the shadow of a rotating spoked wheel activated by a tuning-fork.

The current is obtained by applying electrodes to both forearms and to the calf of the left leg. Electrodes are made of zinc or copper plated with silver. Several varieties are in use (Figs. 8, 9); the shallow pans used in the Mayo Clinic are very convenient for routine clinical work with ambulatory patients. Cohn has recently described a new electrode, simple in construction, consisting of a strip of lead foil 7.5 cm. wide and 22 cm. long, and a

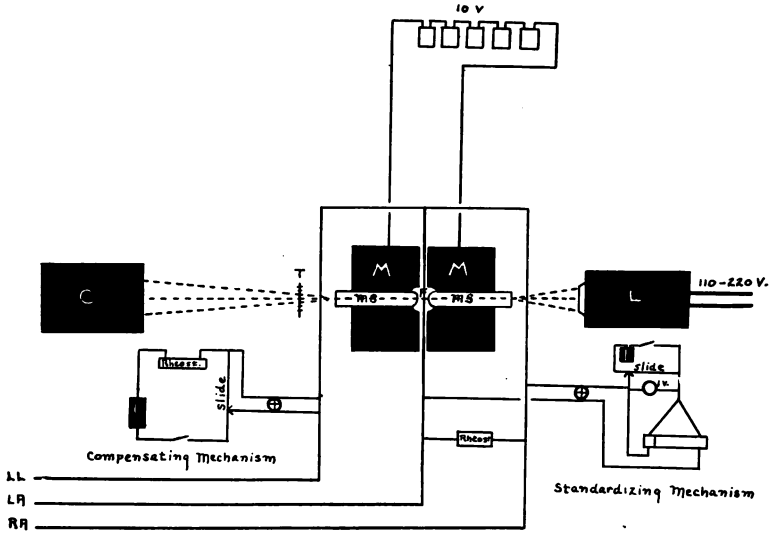

Fig. 7.—Essential wiring of the electrocardiograph: *M*, Electromagnets; *L*, lantern; *C*, camera; *T*, time marker; *LL*, left leg; *LA*, left arm; *RA*, right arm; *F*, fiber; *MS*, microscope.

strip of rubber sheet 9 cm. wide and 30 cm. long. The strips are fastened together near one end by a brass screw, which also carries the binding-post.

The electrolyte is a 20 per cent. saline solution used as warm as can comfortably be tolerated by the patient.

The current represents not only the cardiac action currents but also the difference of potential between the parts of the body to which the electrodes are applied; application of the electrodes

is dependent on local conditions of the skin and tissues. On this account it is important to have the skin of the contact areas thor-

Fig. 8.—Shallow pan electrodes used in the Mayo Clinic.

oughly scrubbed with soap and water, and to use acetone, if necessary, to remove the skin oils. To neutralize this adventitious

Fig. 9.—Flexible electrodes which are bandaged to contact areas.

potential a battery current, exactly equal to it but opposite in direction, is passed through the galvanometer.

DETAILS OF TAKING AN ELECTROCARDIOGRAM

In order to give a clear understanding of the technical details of electrocardiography the procedures step by step are considered:

The electrodes are applied to both forearms and to the calf of the left leg through contact with a felt pad which has been immersed in the warm electrolyte. The sitting posture is very convenient for ambulatory patients, with the electrode pans on

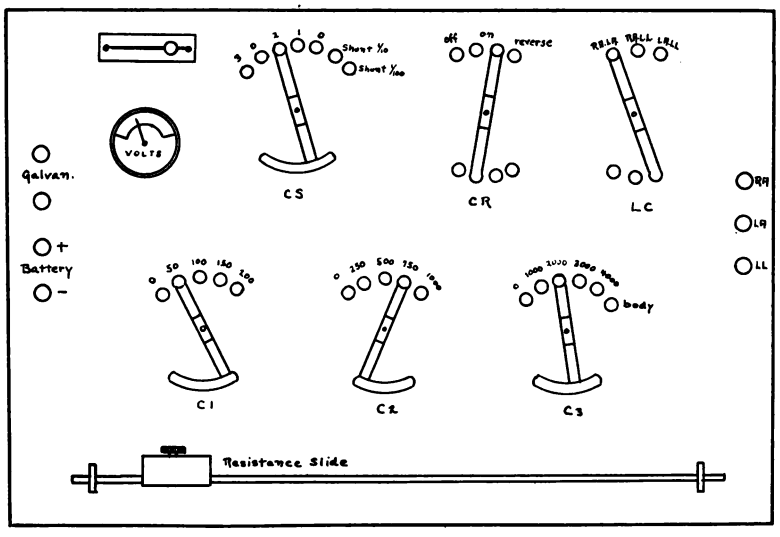

Fig. 10.—Control board of the Cambridge electrocardiograph: *CS*, Current standardizing; *CR*, current reverse; *LC*, derivation control; C_1, C_2, C_3, compensating resistance.

adjustable standards to meet the requirements of the individual patient. The electrodes are directly connected with the galvanometer (Fig. 7).

The control board is that of the Cambridge electrocardiograph (Fig. 10). Figure 11 represents the control board of the Hindle electrocardiograph. The light is turned on, the room darkened, and the fiber shadow focused on the camera mechanism by means of a focusing adjustment attached to the microscope.

Standardization of the fiber tension is necessary to the making of uniform electrocardiograms. The usual tension employed permits

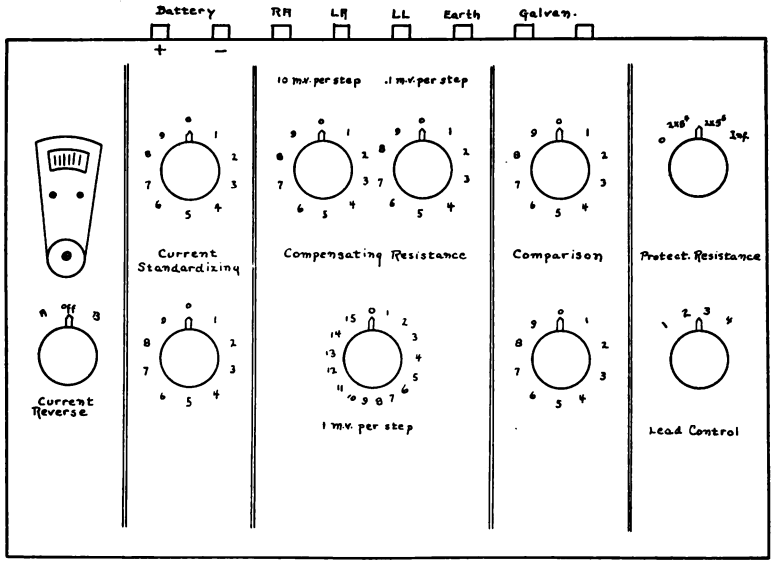

Fig. 11.—Control board of the Hindle electrocardiograph.

an excursion of 1 cm. for each millivolt (Fig. 12). The milled screw at the upper portion of the fiber case controls tension, clock-

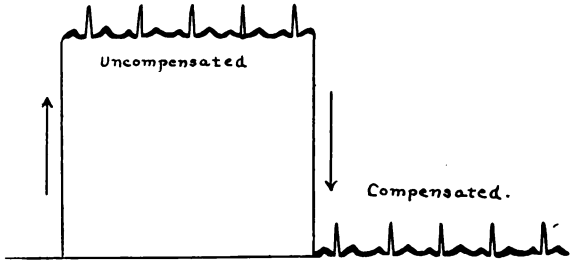

Fig. 12.—Relation between compensated and uncompensated current. (Modified from Hirschfelder.)

wise rotation decreases, while counter-clock-wise rotation increases the tension of the fiber. In order to determine the fiber tension

the magnetic field is excited by closing a switch on the power control board. Movement of the key CS (Fig. 10) deflects the fiber shadow, and movement from 0 to 3 indicates the introduction of 3 millivolts of current; the fiber tension is then adjusted so that the shadow is deflected 3 cm. on the scale just over the aperture of the camera. An excursion of 3 cm. for 3 millivolts is obviously the equivalent of 1 cm. for 1 millivolt.

The next procedure involves the manipulation of key C_3 to contact marked "body"; this brings the patient into the circuit, and the fiber shadow is now found to deflect in rhythm with the patient's cardiac action.

The fiber tension must be standardized again. The fiber shadow is brought back to the center of the field or to 0 by manipulating the resistance slide. If this procedure fails to center the shadow, the key CR is moved to the contact "reverse," and the slide returns the shadow to the desired point.

It is necessary to determine the amount of current introduced to compensate for the resistance encountered through local conditions at the contact areas. After the fiber tension is standardized with the patient in the circuit the key C_3 is moved to 0, and the patient is shunted out of the circuit. The key CS is moved to 3, and the fiber shadow is deflected to a variable point to the right or to the left of 0 on the scale. Keys C_2 and C_1 are manipulated until the fiber shadow is brought back to 3 cm., the standard on the scale. The ohms of resistance are determined by the positions of the keys C_2 and C_1.

The key CS is brought back to 0 and the patient again is introduced into the circuit by moving key C_3 to "body." The fiber shadow is again carefully focused and photographed on the moving bromid paper.

The contacts for the three derivations are controlled by key LC.

The camera mechanism consists of an absolutely dark box and a moving film which is controlled either by the release of an oil cylinder or by a motor rotation arrangement. The speed of the moving film can be regulated accurately.

The coarse and fine ordinate markings of the electrocardiograms indicating fifths and twenty-fifths of a second result from the shadow of the rotating spoked wheel. The horizontal markings indicating millivolts and placed 1 mm. apart are produced by etched lines in the camera lens.

The shadow is again carefully focused and then photographed on the moving bromid paper.

DEVELOPING AND PRINTING THE BROMID PAPER

After the bromid paper has been exposed it is placed in a light-tight container and is ready for developing. The following formula is very satisfactory and produces a print of good contrast:

Developer:

Sodium sulfite (dry)	oz. s̈s̈
Mono-methyl-para-amido-phenol sulfate	gr. xiv
Hydrochinon	gr. lx
Sodium carbonate (dry)	oz. s̈s̈
Water	oz. xx

After developing is complete the print is at once placed into a fixing and hardening bath, where it is allowed to remain for about ten minutes. For the fixing bath Solutions I and II are mixed.

Solution I:

Hyposulfite	oz. xvj
Water	oz. lxiv

Solution II:

Sodium sulfite (dry)	oz. s̈s̈
Pulverized alum	oz. s̈s̈
Commercial acetic acid (28 per cent.)	oz. iij
Water	oz. v

When the print is thoroughly dry it is mounted on light cardboard and may be preserved as a permanent record. A very

convenient size of mount is that used in the Mayo Clinic, 6.5 inches wide and 9.25 inches long. This size permits recording the electrocardiographic diagnosis and a summary of clinical notes.

DERIVATIONS OR LEADS

The action currents are derived from the patient by applying electrodes to both forearms and to the calf of the left leg. The

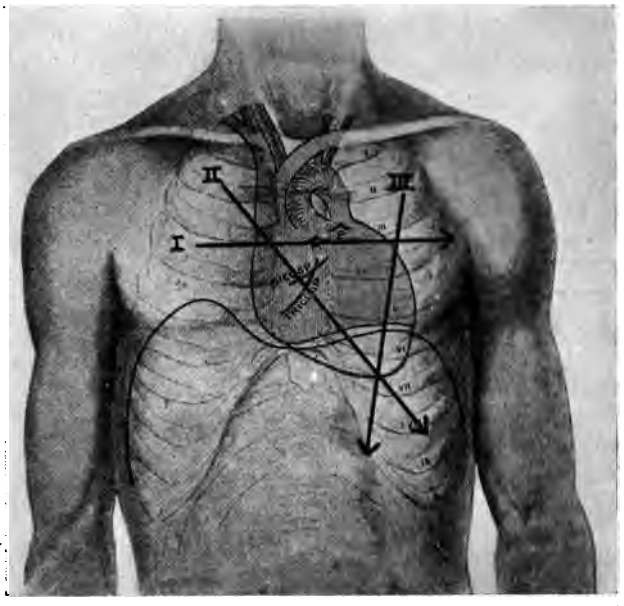

Fig. 13.—The three derivations.

electrodes are paired so that any two conduct the current. The current from the right arm and left arm, derived largely from the base of the heart, constitutes Derivation I (Fig. 13). The current from the right arm and the left leg, corresponding essentially to the long axis of the heart, is Derivation II. The current from the left arm and the left leg, approximating largely the left side of the heart, constitutes Derivation III.

The contracting heart is an area of electropotential and the derivations may be considered planes transecting this electric field. Electrocardiography is a study of the direction, amplitude, and time of the cardiac action currents expressed graphically.

DEVELOPMENT OF MONOPHASIC AND DIPHASIC CURVES

A strip of muscle with parallel fibers (M, Fig. 14) is connected by a non-polarizable electrode (E_1) to a galvanometer (G). The other electrode (E_2) is grounded so that it remains constant. The

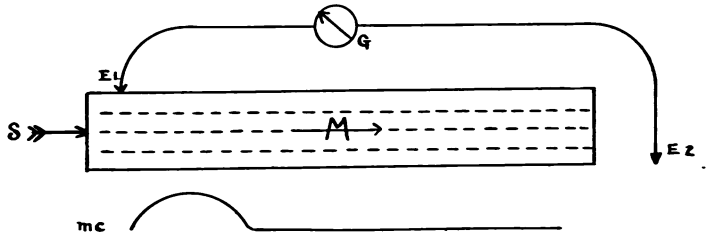

Fig. 14.—Development of the monophasic curve: *M*, Muscle with parallel fibers; *S*, point of stimulation; E_1, non-polarizable electrode; E_2, grounded electrode; *G*, galvanometer; *MC*, monophasic curve. (Modified from Kraus and Nicolai.)

muscle is stimulated at the point S and, as it contracts, this portion becomes electronegative to the remainder of the muscle. The current passes on and the region stimulated first becomes inactive; the electropotential falls and reaches 0. Thus the galvanometer needle is deflected and then comes to rest; if this movement is photographed, the record reveals a monophasic curve.

If the electrode E_2, instead of being grounded, is connected to the other end of the muscle (Fig. 15), and the muscle is stimulated at the point S, this area in contracting becomes electronegative to the resting portions, which are relatively positive. The current moves from —E to $+E_2$ and causes a current to flow through the galvanometer. When the contraction wave reaches the center of the muscle, the current ceases to flow as the positive and negative

ions are neutralized. When this status is reached the galvanometer needle ceases to deflect, but when the contraction wave spreads toward the unstimulated end of the muscle it is again deflected, but in the opposite direction. This end of the muscle is now electronegative to the other as the current flows from $-E_2$ to $+E_1$. In this manner a diphasic curve is produced.

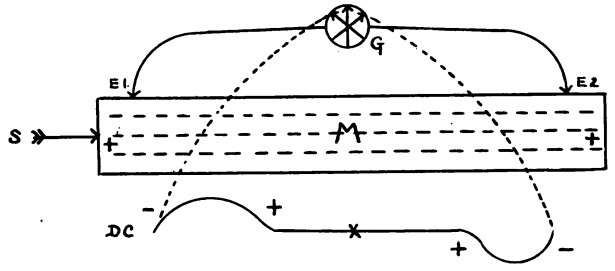

Fig. 15.—Development of the diphasic curve: *M*, Muscle with parallel fibers; *S*, point of stimulation; E_1, E_2, non-polarizable electrodes; *G*, galvanometer; *DC*, diphasic curve. (Modified from Kraus and Nicolai.)

Electrocardiography, as I have emphasized, is dependent on the development of action currents resulting from the heart's activity. This activity (electropotential) is embodied in the muscle mass of the heart; it is obviously modified by the axis of this mass, and by the point of origin and direction of the excitation.

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CHAPTER III

THE NORMAL ELECTROCARDIOGRAM

THE normal electrocardiogram consists of a series of waves or deflections which have been arbitrarily termed P, Q, R, S, T, and U (Fig. 16). The deflections are grouped according to their occurrence in the cardiac cycle; thus P is known as the auricular complex, and Q, R, S, and T as the ventricular complexes. The deflections Q, S, and U are inconstant phenomena; the latter, when present, closely follows the T wave.

A review of the literature on electrocardiography, both experimental and clinical, reveals a variance in views with regard to the

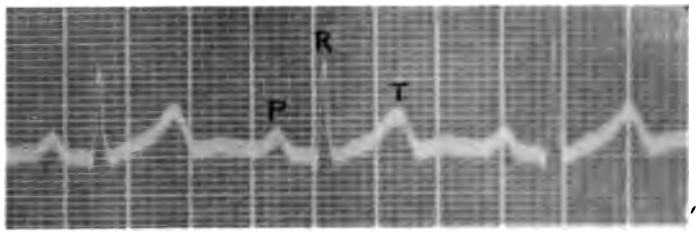

Fig. 16.—Normal electrocardiogram of Derivation II.

interpretation of the normal electrocardiogram. The views that have gained broadest recognition are: (1) all waves are manifestations of excitation and contraction of heart muscle, and (2) the waves result from electric changes accompanying conduction of the impulse and contraction of the muscle.

Einthoven[3] assumes that the right ventricle represents the cardiac base and the left ventricle the apex, and that the dominance of negativity in the right ventricle causes an upward deflection of the galvanometer, while dominance in the left causes a down-

ward deflection of the galvanometer. Thus the R wave is ascribed to contraction of the right heart, the S wave to contraction of the left heart, and the horizontal S–T interval to neutralization of basal and apical negativity. The T wave represents contraction of the right ventricular base outlasting that of the left.

Eppinger and Rothberger object to Einthoven's assumption in ascribing the rôle of cardiac base to the relatively weak right ventricle and of regarding the left ventricle with its massive muscle bulk as the apex.

The views of Kraus and Nicolai are based on the structural arrangement of the ventricular musculature into systems. Following auricular contraction the impulse passes through the auriculoventricular bundle and its contiguous structures. The long P–R interval is explained by slow conduction and the absence of appreciable action currents at this time due to the small muscle mass. The R wave is ascribed to primary activity of the basal portions of the papillary muscles. As the excitation wave spreads toward the apex, the termination of the R wave occurs. The S–T interval is explained by the absence of potential between base and apex. The T wave results from late return of negativity to the base. Einthoven and Kraus and Nicolai agree in general in emphasizing the antagonistic action of electric potential between base and apex.

In distinct opposition to the foregoing views Hoffmann concludes that the electrocardiogram results from two actions, impulse conduction and muscle contraction; that the Q R S complex results from the passage of the impulse through the ventricular conduction system; and that the S–T interval and the T wave result from electropotential caused by the contracting ventricle. Hoffmann produced standstill of the frog's heart by the application of muscarin; he obtained simultaneous electrocardiographic and ventricular suspension curves, and found that the electrocardiogram of the non-beating heart differs from the normal only in the

absence of the T wave. When the muscarin effect was abolished by atropin and the beats returned, the T wave reappeared.

Eyster and Meek, as a result of their experimental work on the relation of the line of isopotential to the formation of the electrocardiogram, and of their critical review of the literature, in general agree with Hoffmann's theory. They believe the R wave to be concerned with conduction, but they do not designate definite structures as conducting mediums. The T wave is the expression of preponderance of contraction on one side of the line of equipotential. Eyster and Meek further show the differences between physiologic curves of conduction and contraction. If a nerve is stimulated where conduction alone occurs, a single monophasic or diphasic electric response occurs. In skeletal muscle this rapid electric change is followed by a slower and more prolonged electric variation. This conforms with the general contour of the electrocardiographic deflections; the R wave is abrupt and steep, the T wave blunt and prolonged.

RELATION OF CARDIAC EVENTS AND THE ELECTROCARDIOGRAM

The relationship of the waves of the electrocardiogram to definitely known cardiac events strongly supports the "conduction-contraction" theory (Fig. 17). By a consideration of accepted relationships of the normal heart sounds a working basis for comparison is established.

Einthoven, Flohil, and Battaerd have shown that the first sound begins at the initiation of ventricular systole and lasts from 0.07 to 0.10 second, is followed by a pause varying from 0.15 to 0.25 second, and is then succeeded by the second sound. These observations have been confirmed by Barker, Frank and Hess, and Weiss and Joachim. Wiggers[18] has shown that the second sound follows closely on the closure of the semilunar valves. Kahn's[10] investigations have demonstrated that the second sound

begins simultaneously with the rise of intraventricular pressure, while Weber and Wirth have demonstrated that it gains its maximum intensity during this period. Piper and Wiggers[19] have shown clearly that the second sound occurs as a diastolic event.

Kahn[11] has further demonstrated that the first sound falls in the pause between the R wave and the T wave, and begins

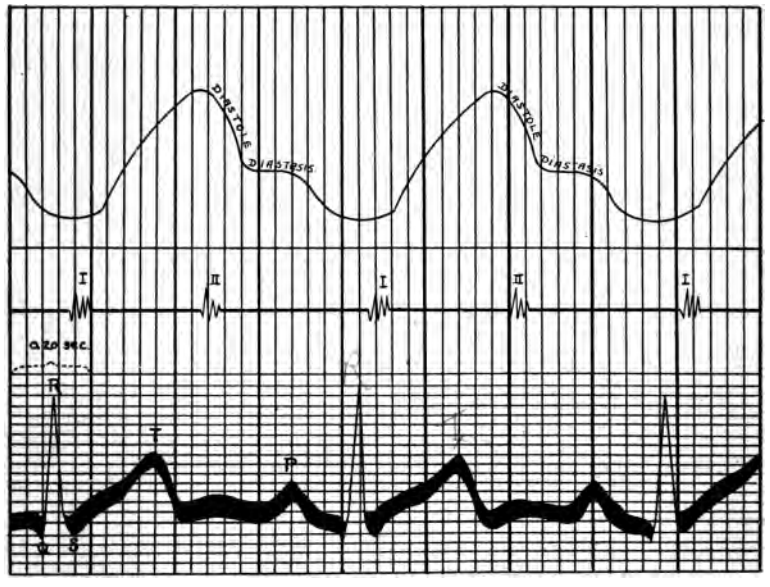

Fig. 17.—Schematic representation showing relationship of the waves of the electrocardiogram to the heart sounds and the ventricular volume curve.

at the moment the R wave disappears and a short time before the rise of the T wave. The second sound begins 0.05 second after the end of the T wave. This relationship reveals the fact that the R wave is completed before ventricular contraction begins and indicates conduction rather than contraction. The T wave definitely occupies the period associated with actual ventricular contraction. It must be recognized, however, that the graphic repre-

sentation of contraction in the electrocardiogram is the expression of changes in electropotential and not the translation of actual contraction.

Significance of the Waves of the Electrocardiogram.—It is evident that variance in views exists with regard to the cause of the individual waves of the electrocardiogram, but after careful analysis of the facts at hand the "conduction-contraction" theory seems the most tenable.

The P wave is evidently a manifestation of both conduction and contraction, indicating conduction of the excitation wave through the sino-auricular node and through the auricle, and associated with actual contraction of the auricle. Eyster and Meek have shown that the period of conduction from the sino-auricular node to the auriculoventricular node is very rapid, averaging 0.023 second in the dog heart. The absence of the P wave in auricular fibrillation, a condition in which auricular contraction has ceased, is positive proof of its association with contraction.

The Q R S complex is evidently a conduction phenomenon and indicates a spread of the excitation wave through the ventricles. The T wave results from contraction of the ventricles, and, as Eyster and Meek have expressed it, represents changes in contraction preponderance on one side of the line of equipotential.

Description of the Individual Waves of the Electrocardiogram.—The P wave is small, fairly abrupt, but has a slightly rounded apex, which normally is always directed upward (positive). It has a deflection amplitude of from 2 to 4 millivolts. This wave is closely followed by the Q R S complex. The P–R interval, the distance from the beginning of the P wave to the beginning of the R wave, normally occupies 0.14 to 0.20 second.

The deflections Q and S, when present, are short, abrupt peaks directed downward (negative) and blend with the ascending and

descending limbs of the R wave. The R wave is an abrupt peaked wave of relatively high amplitude, varying from 10 to 15 millivolts. The base width of this wave normally does not exceed 0.10 second. Because of the extremely rapid deflection of the galvanometer fiber the R wave appears as a delicate line.

The R wave is followed by the T wave, usually rather rounded, with an amplitude of from 3 to 7 millivolts. The S–T interval, which is the distance between the S wave, or the termination of the descending limb of the R wave, when the former is absent, to the end of the T wave, has been shown by Meakins not to exceed 0.28 second. Recently Buchanan in studying the 1028 electrocardiograms of 924 patients in the clinic concluded that the normal S–T interval occupies 0.24 to 0.28 second.

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CHAPTER IV

MATHEMATIC BASIS OF ELECTROCARDIOGRAPHY

EINTHOVEN has shown that the derivations of the electrocardiogram bear a definite mathematic relationship to one another. He assumes that the distribution of potential of the body surface, when an electric tension is present in the heart, is similar to the distribution of potential in an equilateral triangle of good conduction when a potential difference has been developed near its geometric center. The potential differences between the right leg and the left leg were found to be so small as to be negligible in electrocardiography, and the two legs could be regarded as one iso-electric point.

The three derivations form the sides of an equilateral triangle (Fig. 18). By the use of this scheme it is possible to determine the direction of the potential difference in the heart at any given instant and to determine a derived value of the electromotive force in the heart, which is the manifest value, bearing a constant relationship to the actual potential difference in the heart. Einthoven defines manifest value as that dimension in millivolts which is derived when the electric axis and derivations coincide. Thus, he differentiates the actual size of the deflections as they are reproduced in the electrocardiogram, and their manifest size or difference of electropotential. The manifest value varies in direct ratio to the changes in magnitude of the actual potential differences produced during each heart cycle.

The string galvanometer is an instrument which measures differences of potential, and because the resistance in its circuit is not infinite there is a slight tendency to decrease the difference

of potential between the contact points (derivations), but this difference is so small that it falls within the limits of experimental error.

The height of the ordinate in Derivation II of the electrocardiogram at any instant is the sum of the heights of the ordinates in Derivations I and III; Derivation II equals Derivation I plus Derivation III. If it is desired to measure the difference of potential of Derivation II at any instant, this can be accomplished by con-

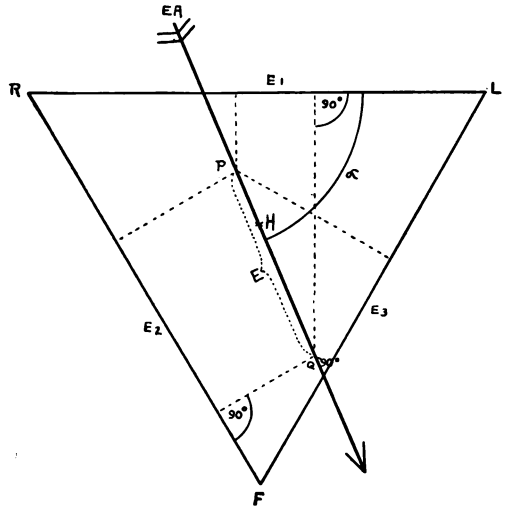

Fig. 18.—Equilateral triangle using polar system of co-ordinates. (After Einthoven, Fahr, and De Waart.)

necting the galvanometer with the right arm and the left leg. This potential difference can also be determined indirectly by obtaining the differences of Derivation I and Derivation III and adding them. Therefore, if the electrocardiogram elicits accurate value of potential differences, Derivation II = Derivation I + Derivation III at any and every instant during the heart cycle. Expressed in another way, Derivation II − Derivation I = Derivation III

Einthoven used the polar system of co-ordinates in his computations. In Fig. 18 the right and left arms and the left foot are designated R, L, F. They form the angles of the equilateral triangle. The point H is the center and represents the heart. The arrow (E A) represents any given electric axis; the angle it forms with Derivation I (RL) is represented by α, and any given length PQ may be designated E; the right angle projection of its length will give its corresponding value in the various derivations, so that in Derivation I the projection PQ equals E_1, in Derivation II, E_2, and in Derivation III, E_3. The distances E_1, E_2, and E_3 are proportional, so that $E_1 : E_2 : E_3$. Since any angle of an equilateral triangle is equal to 60°, the following trigonometric formulas are derived:

$$E_1 = E \cos \alpha$$
$$E_2 = E \cos (\alpha - 60°)$$
$$E_3 = E \cos (120° - \alpha)$$
$$E_3 = E_2 - E_1$$

The determination of the angle formed by the electric axis and the derivations is shown in the foregoing scheme. For clinical purposes only those angles which are multiples of 30° are used, the smaller angles being discarded, thereby simplifying the computation. For example, when

$\alpha = 0°$, then $E_1 : E_2 : E_3 = 1.0 : +0.5 : -0.5$
$\alpha = 30°$, then $E_1 : E_2 : E_3 = 1.0 : 1.0 : 0$
$\alpha = 60°$, then $E_1 : E_2 : E_3 = +0.5 : 1.0 : -0.5$
$\alpha = 90°$, then $E_1 : E_2 : E_3 = 0 : 1.0 : 1.0$

It is necessary to measure the deflections of the electrocardiogram at identical instants of the heart cycle.

From these mathematic proportions, the angle α can be derived within 30°. The manifest value = the actual size of R_1 (E_1) when $\alpha = 0°$; of R_2 (E_2) when $\alpha = 60°$; and of R_3 (E_3) when $\alpha = 120°$. Table 1 shows values for approximation within 10°, and Table 2

interpolation values whereby the exact angle may be determined. If, for example, $R_1 = 3.8$, $R_2 = 12.5$, and $R_3 = 8.5$, R_1 (E_1) : R_2 (E_2) : R_3 (E_3) = 3.8 : 12.5 : 8.5.

Table 1*

Angle Degrees	Registered potential differences E			Manifest potential differences
	E_1	E_2	E_3	E
0	10.0	5.0	-5.0	10.0
10	10.0	6.5	-3.5	10.2
20	10.0	8.2	-1.8	10.7
30	10.0	10.0	0.0	11.5
40	8.2	10.0	1.8	10.7
50	6.5	10.0	3.5	10.2
60	5.0	10.0	5.0	10.0
70	3.5	10.0	6.5	10.2
80	1.8	10.0	8.2	10.7
90	0.0	10.0	10.0	11.5
100	-1.8	8.2	10.0	10.7
110	-3.5	6.5	10.0	10.2
120	-5.0	5.0	10.0	10.0
130	-6.5	3.5	10.0	10.2
140	-8.2	1.8	10.0	10.7
150	-10.0	0.0	10.0	11.5
160	-10.0	-1.8	8.2	10.7
170	-10.0	-3.5	6.5	10.2
+180	-10.0	-5.0	5.0	10.0
-170	-10.0	-6.5	3.5	10.2
-160	-10.0	-8.2	1.8	10.7
-150	-10.0	-10.0	0.0	11.5
-140	-8.2	-10.0	-1.8	10.7
-130	-6.5	-10.0	-3.5	10.2
-120	-5.0	-10.0	-5.0	10.0
-110	-3.5	-10.0	-6.5	10.2
-100	-1.8	-10.0	-8.2	10.7
-90	0.0	-10.0	-10.0	11.5
-80	1.8	-8.2	-10.0	10.7
-70	3.5	-6.5	-10.0	10.2
-60	5.0	-5.0	-10.0	10.0
-50	6.5	-3.5	-10.0	10.2
-40	8.2	-1.8	-10.0	10.7
-30	10.0	0.0	-10.0	11.5
-20	10.0	1.8	-8.2	10.7
-10	10.0	3.5	-6.5	10.2
0	10.0	5.0	-5.0	10.0

* After Einthoven

To derive an approximate value within 10° (Table 1), 12.5, the tallest deflection, becomes the denominator, for example, $\frac{10}{12.5}$ = 0.8. This fraction is then substituted in the equatio-

$E_2 : E_3 = 3.0 : 10.0 : 6.8$. In Table 1 the angle α falls between 70 and 80°, and E_1 between 70 and 80° has a value 3.5 and 1.8, a difference of 1.7. In the interpolation table the nearest value to this difference is 1.8, which is equivalent to 10°, so that $\alpha = 70° + 10° = 80°$. By this same process the other deflections may be introduced into the computation. After the angle α has been

Table 2*

INTERPOLATION

Derivations	Differences in degrees	Potential differences +E	Differences in degrees	E
I	0	0	10	11.5
	2	0.4	8	11.3
	4	0.8	6	11.1
	6	1.2	4	11.0
	8	1.5	2	10.8
	10	1.8	0	10.7
II	2	2.2	8	10.5
	4	2.5	6	10.4
	6	2.9	4	10.3
	8	3.2	2	10.2
	10	3.5	0	10.2
III	2	3.8	8	10.1
	4	4.1	6	10.1
	6	4.4	4	10.0
	8	4.7	2	10.0
	10	5.0	0	10.0

* After Einthoven

determined it is possible to determine the manifest size of the various deflections by trigonometry:

$$E = \frac{E_1}{\cos \alpha}$$

$$E = \frac{E_2}{\cos (\alpha - 60°)}$$

$$E = \frac{E_3}{\cos (120° - \alpha)}$$

Mann recently has suggested the substitution of the rectangular system of co-ordinates for the polar system for the purpose of mathematic simplification. He believes that it is simpler to think

of a point with the value X = 4, Y = 3, than it is to think of the same point with the value E = 5, $\alpha = 37°$.

Figure 19 represents an equilateral triangle in which the rectangular system of co-ordinates is applied. The values of the rectangular co-ordinates (X, Y) of a point (A) are found algebraically in terms of the three derivations (E_1, E_2, and E_3).

PROPOSITION: The longest projection on the sides of an equilateral triangle of any straight line drawn within an equilateral triangle equals the sum of the projections on the other two sides.

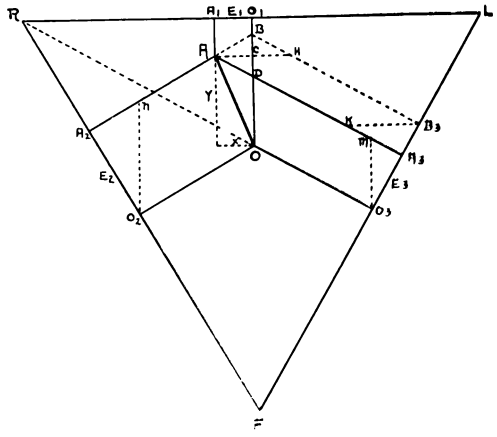

Fig. 19.—Equilateral triangle using rectangular system of co-ordinates. (After Mann.)

LET A O* be any straight line drawn within the equilateral triangle R L F. Let e_1, e_2, and e_3 be the projections of A O on the three sides of the triangle, obtained by dropping perpendiculars (AA_1, AA_2, AA_3, OO_1, OO_2, OO_3) from the ends of the line A O upon the sides of the triangle.

TO PROVE that $e_2 = e_3 + e_1$:

* In the figure the point O has been made the center of the triangle for the sake of simplicity in construction, but the proof does not depend on this fact, and will hold whatever be the position of the line O A. As a matter of fact, the point O is taken at the center of the triangle in the subsequent part of this book

CONSTRUCTION: 1. Produce A_2A until it meets OO_1 at B.

2. Draw BB_3 perpendicular to L F.

3. Draw A H parallel to R L, cutting B O at C.

4. Draw K B_3 parallel to A. H.

5. Draw R O_3* the perpendicular bisector of L F.

PROOF

$A_2O_2 = B_3O_3$ for they are the projections of OB, and the projections on the sides of an isosceles triangle of any line perpendicular to the base are equal.

$A_2O_2 = A_3O_3 + A_3B_3$ for $B_3O_3 = A_3O_3 + A_3B_3$.

$\epsilon_2 = \epsilon_3 + A_3B_3$ for $A_2O_2 =$ and $A_3O_3 = \epsilon_2$.

$= \epsilon_3 + \frac{1}{2}$K B_3 for $A_3B_3 = \frac{1}{2}$ K B_3 because triangle K A_3B_3 is similar to triangle R L O_3 and L $O_3 = \frac{1}{2}$ R L since F L = R L and L $O_3 = \frac{1}{2}$ F L.

$= \epsilon_3 + \frac{1}{2}$A H for K B_3 = A H because parallel lines included between parallel lines are equal.

$= \epsilon_3 +$ A C for $\frac{1}{2}$ A H = A C because A C = C H, since triangle A B C = triangle H B C. (The two triangles have the common side B C, angle A C B = angle H B C = a right angle, and angle A B C = angle H B C = 60° since perpendiculars to the sides of an equilateral triangle intersect at angles of 60°.)

$\epsilon_2 = \epsilon_3 + \epsilon_1$ for A C = ϵ_1, because parallel lines included between parallel lines are equal.

Q. E. D.

TO FIND THE VALUE OF THE RECTANGULAR CO-ORDINATES (X, Y) OF THE POINT A IN TERMS OF ϵ_1, ϵ_2, ϵ_3

Draw NO_2 and MO_3 parallel to OO_1

Then X = A C = $A_1O_1 = \epsilon_1$

Y = C O = (B O — B C) = (D O + D C)

$-\frac{1}{2}$ (B O — B C + D O + D C)

$= \frac{1}{2}$ (B O + B O) for B C = D C because triangle A B D is an equilateral triangle (having its three sides perpendicular to the sides of the equilateral triangle R L F) and A C is the perpendicular bisector (being parallel to R L and therefore perpendicular to BD).

$\delta = \frac{1}{2}$ ($NO_2 + MO_3$) for B O = N O_2 and D O = M O_3 because parallel lines included between parallel lines are equal.

$= \frac{1}{2}\left(\epsilon_2 \frac{2}{\sqrt{3}} + \epsilon_3 \frac{2}{\sqrt{3}}\right)$ for angle N O_2A_2 = 30° = angle M O_3A_3 and the secant of 30° = $\frac{2}{\sqrt{3}}$.

$Y = \frac{\epsilon_2 + \epsilon_3}{\sqrt{3}}$.

Table 3*

$\sqrt{3} = 1.7320+$

$\frac{E_2 + E_3}{\sqrt{3}}$	0	1	2	3	4	5	6	7	8	9	Differences
0		0.0577	0.1155	0.1732	0.2309	0.2887	0.3464	0.4041	0.4619	0.5196	
1	0.5773	0.6351	0.6928	0.7506	0.8083	0.8660	0.9238	0.9815	1.039	1.097	1. 0.00577
2	1.1547	1.212	1.270	1.328	1.386	1.443	1.501	1.559	1.617	1.674	2. 0.01155
3	1.7321	1.790	1.848	1.905	1.963	2.021	2.079	2.136	2.194	2.252	3. 0.01732
4	2.3094	2.367	2.425	2.483	2.540	2.598	2.656	2.714	2.771	2.829	4. 0.02309
5	2.8868	2.944	3.002	3.060	3.118	3.175	3.233	3.291	3.349	3.406	5. 0.02887
6	3.4441	3.522	3.580	3.631	3.695	3.753	3.811	3.868	3.926	3.984	6. 0.03464
7	4.0415	4.099	4.157	4.215	4.272	4.330	4.388	4.446	4.503	4.561	7. 0.04041
8	4.5188	4.677	4.734	4.791	4.850	4.907	4.965	5.023	5.081	5.138	8. 0.04619
9	5.1962	5.254	5.312	5.369	5.427	5.485	5.543	5.600	5.658	5.716	9. 0.05196

*After Mann.

In obtaining the deflection values they must be arranged so that at any instant during the heart cycle, Derivation II – Derivation I = Derivation III.

The value of X for each moment (0.01 second) is known, as is

Table 4*

	Derivation I.	Derivation II.	Derivation III	Y $\frac{2+3}{\sqrt{3}}$
0	0.0	0.0	0.0	0.0
1	0.1	0.1	0.0	0.06
2	0.4	0.4	0.0	0.2
3	0.7	0.7	0.0	0.4
4	1.0	1.0	0.0	0.6
5	1.3	1.3	0.0	0.75
6	1.0	1.0	0.0	0.6
7	0.8	0.8	0.0	0.46
8	0.5	0.4	-0.1	0.17
9	0.1	-0.2	-0.3	-0.3
10	-0.3	-0.4	-0.1	-0.3
11	-0.3	-0.3	0.0	-0.17
12	-0.3	-0.3	0.0	-0.17
13	-0.3	-0.3	0.0	-0.17
14	-0.3	-0.3	0.0	-0.17
15	-0.3	-0.3	0.0	-0.17
16	-0.3	-0.3	0.0	-0.17
17	-0.3	-0.3	0.0	-0.17
18	-0.3	-0.3	0.0	-0.17
19	0.0	0.4	0.4	0.46
20	10.5	9.7	-0.8	5.1
21	8.2	12.0	3.8	9.3
22	5.0	8.0	3.0	6.4
23	0.0	2.0	2.0	2.3
24	-0.5	-0.2	0.3	0.06
25	-0.3	-0.3	0.0	-0.17
26	-0.3	-0.3	0.0	-0.17
27	-0.3	-0.3	0.0	-0.17
28	-0.2	-0.2	0.0	-0.1
29	-0.1	-0.1	0.0	-0.06
30	0.0	0.0	0.0	0.0
31	0.1	0.1	0.0	0.06
32	0.2	0.2	0.0	0.1
33	0.4	0.4	0.0	0.2
34	0.5	0.5	0.0	0.3
35	0.6	0.6	0.0	0.35
36	0.7	0.7	0.0	0.4
37	0.7	0.7	0.0	0.4
38	0.8	0.8	0.0	0.46
39	0.8	0.8	0.0	0.46
40	0.9	0.9	0.0	0.5
41	0.9	0.9	0.0	0.5
42	0.9	0.8	-0.1	0.4
43	0.9	0.6	-0.3	0.17
44	0.8	0.3	-0.5	-0.1
45	0.7	0.2	-0.5	-0.17
46	0.5	0.1	-0.4	-0.17
47	0.3	0.0	-0.3	-0.17
48	0.2	0.0	-0.2	-0.1
49	0.1	0.0	-0.1	-0.06
50	0.0	0.0	0.0	0.0

*After Mann

the value of Derivation I, but the value of Y must be determined by dividing the algebraic sum of Derivations II and III by $\sqrt{3}$. This is done by means of Table 4, which is used like a logarithm table, successively giving the value of Y.

For example: At the instant noted 20 in Table 4, the ordinate in Derivation II measured 9.7 mm., and the ordinate in Derivation III measured −0.8 mm. The algebraic sum of 9.7 and −0.8 is 8.9. If this number is located in Table 3 it is found by tracing the horizontal row marked 8 to its intersection with the vertical row 9, the value 5.138 is obtained. Thus the value for Y is approximately 5.1. The values for Y at other instants are found in the same manner by adding the values of Derivations II and III and using the table to simplify the process of dividing by $\sqrt{3}$.

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CHAPTER V

CARDIAC ARHYTHMIAS

MUCH of our knowledge regarding cardiac arhythmia has been gained by means of graphic methods of precision, particularly with the polygraph and the electrocardiograph. Interpretation of the graphic records has aided materially in identifying and localizing the abnormalities of the cardiac mechanism underlying arhythmia.

The classification of disorders of the heart's action employed in this work is based on the fundamental alterations of the cardiac mechanism.

CARDIAC ARHYTHMIAS

1. Sinus arhythmia
2. Premature contractions (extrasystoles):
   - (*a*) Auricular
   - (*b*) Nodal (junctional)
   - (*c*) Ventricular
3. Auricular fibrillation
4. Ventricular fibrillation

Arhythmia is often present in auricular flutter and invariably present in complete auriculoventricular dissociation (complete heart-block), but the fundamental underlying disturbances of the cardiac mechanism are not arhythmia. In flutter we are concerned with an ectopic tachycardia, and in complete auriculoventricular dissociation with a disturbance in impulse conduction, the arhythmia being purely a secondary phenomenon. This classification, I believe, is not confusing; its basis reverts directly to the fundamental disturbances of the cardiac mechanism.

SINUS ARHYTHMIA

Sinus or respiratory arhythmia is frequently observed in healthy young persons. Mackenzie has termed it the "youthful type of irregularity." A slight acceleration of rate occurs during inspira-

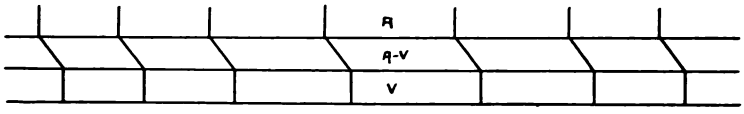

Fig. 20.—Schematic graph of sinus arhythmia: *A*, Auricle; *A-V*, auriculo-ventricular junctional tissues; *V*, ventricle. These abbreviations are employed in the subsequent graphs.

tion and a slight slowing during expiration. The direct cause of the arhythmia is vagus stimulation resulting from respiratory action which affects the sino-auricular node, and causes a transient

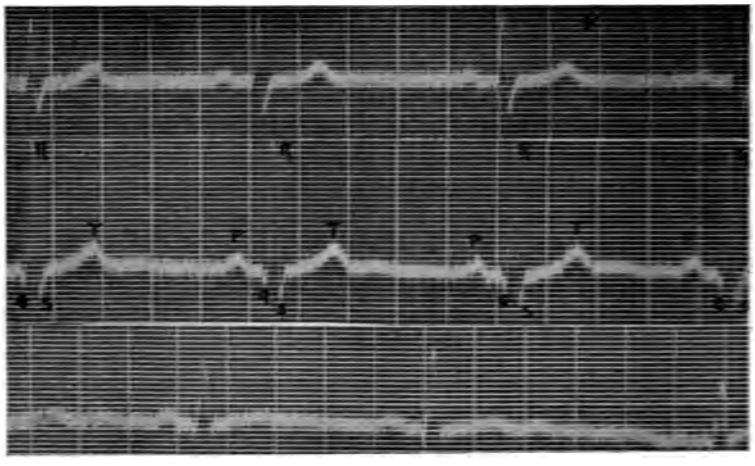

Fig. 21.—Sinus arhythmia. Electrocardiogram in Derivations I, II, and III.

slowing of the entire heart. Sinus arhythmia occurs in individuals whose vagus mechanism is temporarily or permanently unstable, and in whom nervous irritability permits otherwise inadequate stimuli to act. Exertion frequently induces sinus arhythmia.

It must be remembered, however, that sinus arhythmia, *per se*, while not indicative of organic heart disease, may be associated with changes in the heart of a very serious nature. Sinus arhythmia in older patients, with evident degenerative myocardial changes, usually is associated with a persistently slow pulse-rate. Figure 20 represents the character of the arhythmia. Figure 21 is an electrocardiogram of a patient with sinus arhythmia. All complexes are normal in contour and maintain their proper relationship to one another. The arhythmia occurs through variations in the lengths of the cycles resulting from acceleration and slowing of rate.

Respiratory Amplitude Variation.—Another respiratory effect on the cardiac mechanism is seen in Fig. 22. Arhythmia is not

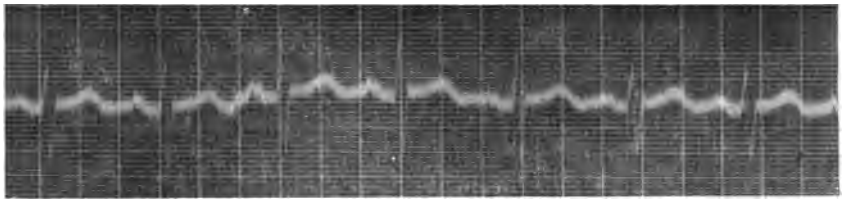

Fig. 22.—Electrocardiogram in Derivation II. Phasic variation with breathing.

present, but a phasic variation in the amplitude of the R wave occurs corresponding to the respiratory cycle. This also is a vagus effect and is the paradox of Riegel's pulse (pulsus paradoxus) of adhesive pericarditis.

PREMATURE CONTRACTIONS (EXTRASYSTOLES)

Premature contractions or extrasystoles constitute one of the most common arhythmias of adult life. They often occur in perfectly normal hearts and in hearts with organic disease. The arhythmia must not be considered indicative of heart disease, but simply the expression of increased cardiac irritability, which may result from functional or organic causes.

Etiology.—The frequency of premature contractions in persons with nervous instability and without demonstrable evidence of organic heart disease definitely establishes a neurogenic origin. The overindulgence in tea, coffee, and tobacco in some persons increases cardiac irritability sufficiently to produce this arhythmia. In persons with organic heart disease premature contractions are frequently observed in the myocardial degeneration associated with hypertension and with coronary sclerosis, and are not infrequent in the senile myocardium.

Experimental.—The application of a single induction shock to any portion of the musculature of the exposed animal heart produces a contraction which interrupts the normal cardiac rhythm by occurring before the next normal beat. The premature beat is followed by a longer pause than normal, since the heart is insensitive to the succeeding sinus stimulus while in the state of contraction.

By ligation of the left descending branch of the coronary artery, and in most instances by impairing the circulation of the right vessel, Lewis quite constantly produced premature contractions which originated in the ventricles.

By the intravenous injection of salts Rothberger and Winterberg produced premature contractions of ventricular origin in dogs. They found that combined stimulation of the vagi and the accelerators caused cessation of the heart-beat, but after the injection of from 5 to 10 mg. of barium chlorid in 1 per cent. aqueous solution premature contractions occurred. Calcium chlorid produced similar results. They concluded that these salts increase the irritability of the ventricles.

Hirschfelder caused premature contractions to arise auricles by experimentally producing stenosis at the au ventricular orifices.

Mechanism.—Premature contractions result from stim

ing at some point in the cardiac musculature away from the normal point of origin, the sino-auricular node. These abnormal beats are termed "ectopic" because of their aberrant location. They may arise in any portion of the cardiac musculature, in either auricle, in the auriculoventricular junctional tissues, or in either ventricle.

The premature beat, as its name implies, occurs before the anticipated normal beat, and is followed by a pause exceeding the length of the normal cycle. This pause is known as the compensatory period and denotes a prolonged refractory phase while the heart is in contraction, and hence insensitive to further stimulation.

Premature contractions may interrupt the normal rhythm irregularly. They may occur regularly every second beat, or every third beat, giving rise to the pulsus bigeminus and pulsus trigeminus. They may be interspersed at greater intervals, occurring every fourth or fifth beat. Again, short series of successive premature contractions may occur. Occasionally, when the heart rate is slow, an ectopic contraction may fall in the place of a normal beat, and arhythmia is not produced. Such a beat is referred to as being interpolated. When cardiac irritability is marked, multiple foci of abnormal stimuli may occur, giving rise to premature contractions in various portions of the heart.

Premature contractions, occurring early in the cardiac cycle, especially if there is a high peripheral resistance, are incapable of opening the aortic cusps, since intracardiac pressure fails to equal the pressure in the aorta, and they are thus not transmitted to the peripheral arteries. Such contractions are termed "abortive" or "frustrane."

Auricular Premature Contractions.—Premature contraction arising near the sino-auricular node gives an electrocardiogram which differs from the normal only in the resulting arhythmia. The P wave occurs prematurely, but is followed by a Q R S group and a T wave of normal characteristics. The impulse responsible

for this ectopic beat is in the immediate vicinity of the normal pace-maker, and, being supraventricular, permits the resulting excitation to traverse normal paths to provoke ventricular response.

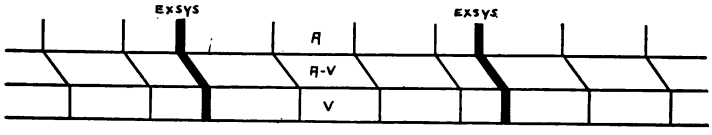

Fig. 23.—Schematic graph of auricular premature contractions.

Figure 23 illustrates the mechanism of auricular premature contractions. Figure 24 is an electrocardiogram of such an auricular premature contraction.

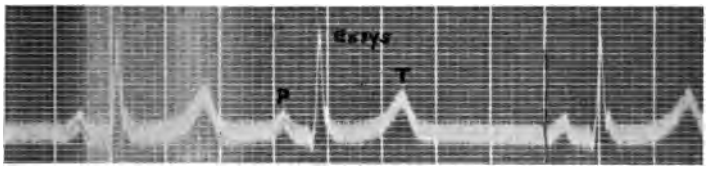

Fig. 24.—Auricular premature contraction (*Exsys.*) arising in the immediate vicinity of the sino-auricular node. The *P* wave is upright. Electrocardiogram in Derivation II.

When the ectopic impulse arises at a point in the auricular musculature distant from the sino-auricular node the P wave

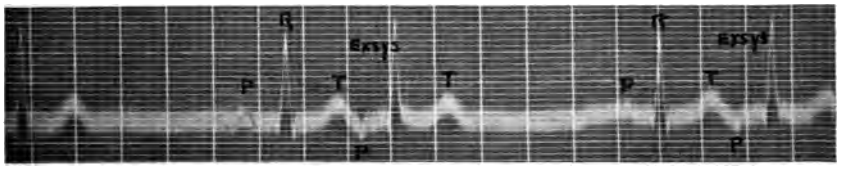

Fig. 25.—Alternating auricular premature contraction (*Exsys.*) arising at a point in the auricular musculature distant from the sino-auricular node. The *P* wave is negative (inverted). Electrocardiogram in Derivation II.

becomes negative (inverted). Proof that the negative P wave is indicative of a change in the pace-maker is found in the work of

Lewis, von Hoesslin, Einthoven, Fahr and de Waart, Wilson, and Carter and Wedd. Figure 25 illustrates this type of premature auricular contraction.

White and Stevens have called attention to the fact that at times premature auricular contractions show aberrant ventricular responses. They showed that the greater the prematurity of the auricular beats, the greater is the likelihood of aberration of the responding ventricular complex. An example of such a case is found in Fig. 26.

Auricular premature contractions are at times observed preceding or following paroxysms of auricular tachycardia, auricular flutter, and auricular fibrillation.

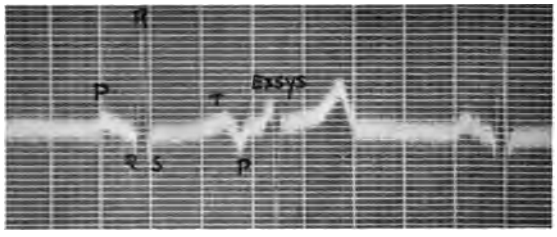

Fig. 26.—Auricular premature contraction with aberrant ventricular response.

Nodal or Junctional Premature Contractions.—Premature contractions arising in the auriculoventricular junctional tissues are referred to as nodal. They may arise in the node or bundle. In the resulting electrocardiograms one or more of several characteristics are present:

1. Owing to the intermediate region of ectopic impulse formation both auricles and ventricles may contract simultaneously. In this status the P wave is not apparent in the abnormal complex, but occurs synchronous with the R wave, giving a greater amplitude to the latter (Fig. 27). In Fig. 28 the absence of the P wave and the increased amplitude of the R wave of the premature complex may be noted. The ventricular complex is usually unaltered,

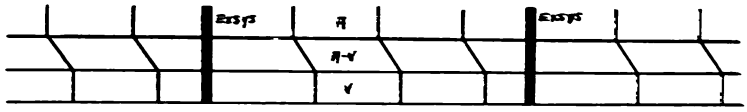

Fig. 27.—Schematic graph of nodal premature contractions. P–R interval 0.

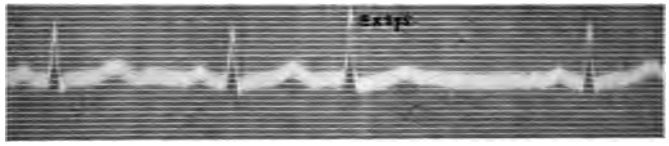

Fig. 28.—Nodal premature contraction. P–R interval 0. Electrocardiogram in Derivation II.

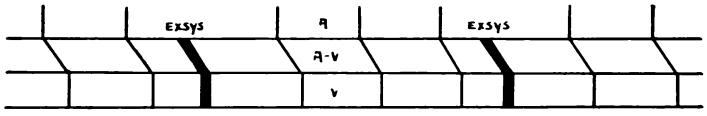

Fig. 29.—Schematic graph of nodal premature contractions. P–R interval diminished.

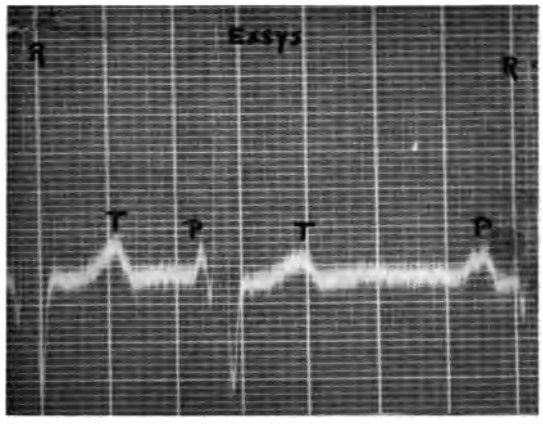

Fig. 30.—Nodal premature contraction (*Exsys.*). Diminished P–R interval, 0.04 second. Electrocardiogram in Derivation II.

as the impulse provokes ventricular contraction after having traversed junctional paths. If the impulse arises very low in the

junctional tissues, however, an aberrant ventricular complex results, very much like that of a premature contraction arising in the ventricles.

2. The auricles may contract just slightly in advance of the ventricles and produce a diminished A_s–V_s conduction time (P–R interval) (Fig. 29). Figure 30 shows this type of nodal premature contraction. The P–R interval is only 0.04 second.

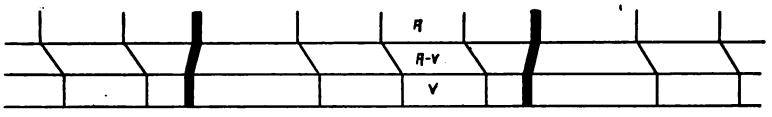

Fig. 31.—Schematic graph of nodal premature contraction. Presence of an R–P interval.

3. Occasionally the ventricles contract before the auricles and the P wave follows instead of preceding the R wave. Under such conditions an R–P interval exists. The ventricular complex usually shows aberration (Fig. 31). Figure 32 shows this type of premature contraction in the electrocardiogram. The ventricular complex Q R S is slightly altered and the P wave is negative.

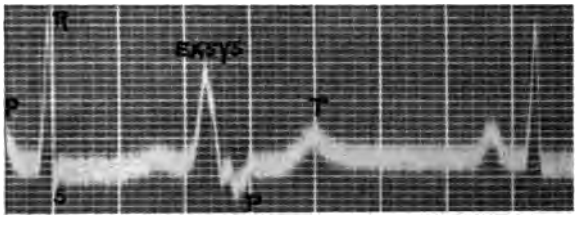

Fig. 32.—Nodal premature contraction (*Exsys.*). Presence of an R–P interval. Electrocardiogram in Derivation II.

Experimental work dealing with nodal premature contractions has been done by Hering, Lewis, Lohmann, and Rothberger and Winterberg. Wilson has studied these premature contractions clinically in conjunction with venous tracings and formulated definite electrocardiographic criteria.

Ventricular Premature Contractions.—Premature contractions of ventricular origin may arise in any portion of the ventricular musculature. Owing to the fact that the resulting excitations

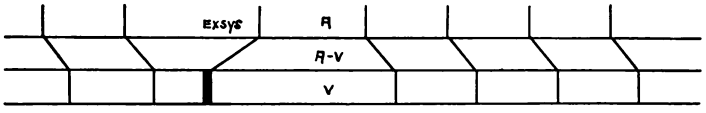

Fig. 33.—Schematic graph of ventricular premature contraction.

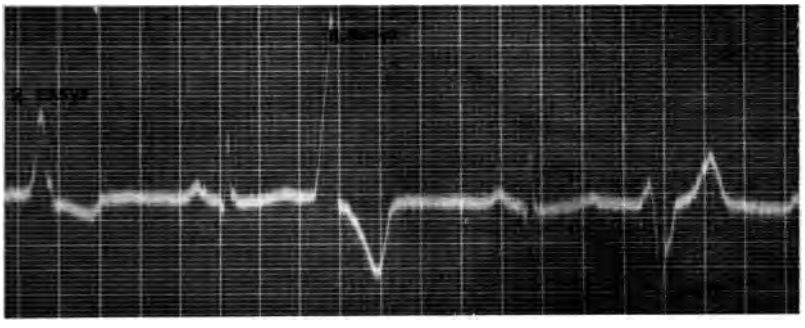

Fig. 34.—Ventricular premature contractions (*Exsys.*) resulting from multiple foci of ectopic stimuli. Premature contractions arising in the right and left ventricles. Electrocardiogram in Derivation II.

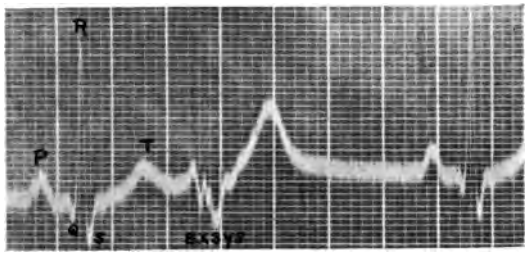

Fig. 35.—Left ventricular premature contraction of low amplitude (*Exsys.*). Electrocardiogram in Derivation II.

follow abnormal paths in the ventricles, the electrocardiograms reveal abnormal ventricular complexes. The Q R S complex is frequently of high amplitude and diphasic, and the contour is

altered by notching of the apex or the complex limbs. The T wave of the abnormal complex is often exaggerated, very peaked, and directed downward (negative). The amplitude of the Q R S complex at times is not exaggerated, and occasionally is of very

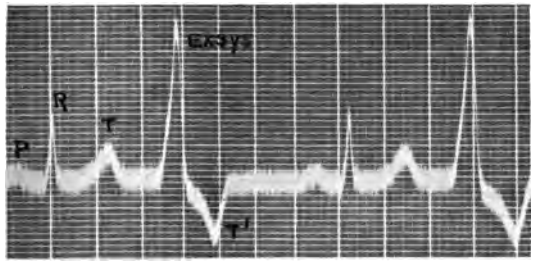

Fig. 36.—Right alternating ventricular premature contractions (*Exsys.*). Electrocardiogram in Derivation II.

low voltage (Fig. 33). Figure 34 is an electrocardiogram in Derivation II showing ventricular premature contractions resulting from multiple foci of ectopic stimuli. The premature contraction arising in the right ventricle has a very large upstroke and a T wave

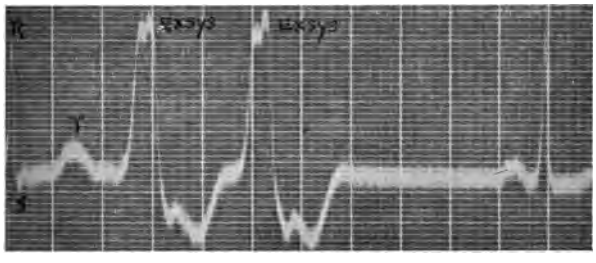

Fig. 37.—Paired ventricular premature contractions. Electrocardiogram in Derivation II.

directed downward, while that arising in the left ventricle presents the mirror image of the right. The P wave is often submerged in the aberrant Q R S complex. Figures 35 to 39 show premature contractions interrupting the normal rhythm in various ways.

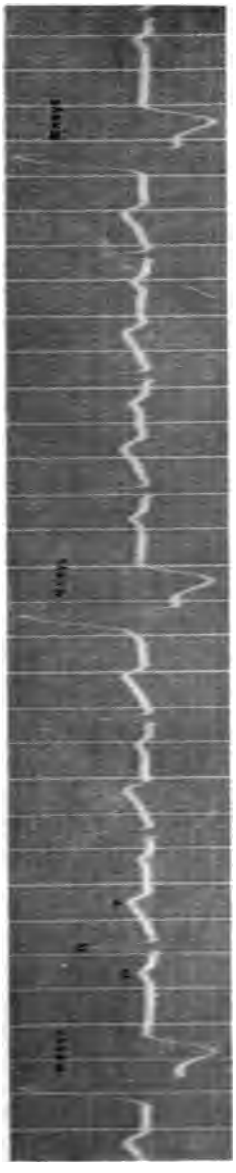

Fig. 38. Ventricular premature contractions occurring regularly every fourth beat. Electrocardiogram in Derivation II.

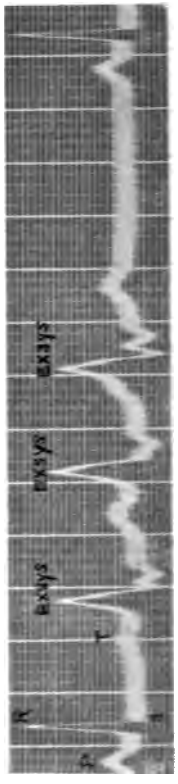

Fig. 39. Short series of nodal premature contractions (*Exsys.*). Electrocardiogram in Derivation II.

AURICULAR FIBRILLATION

The most important and most frequent disorder of cardiac rhythm is that due to auricular fibrillation. As early as 1890 Mackenzie identified the arhythmia of auricular fibrillation as a distinct entity, but he was at a loss to explain the mechanism responsible for the ventricular form of the waves in his venous pulse tracings. In 1902, after carefully analyzing cases that he had observed for a considerable period, he was convinced that this peculiar arhythmia resulted from cessation of auricular function and he believed the auricles to be paralyzed. In Mackenzie's earlier necropsies distended, thin-walled auricles were revealed in several instances, but in his later cases auricular hypertrophy was encountered. Mackenzie, realizing that his previous conception of the mode of production of this arhythmia was not substantiated by the newer findings, argued that auricles and ventricles must be contracting simultaneously, and in 1908 he published his work, referring to the condition as "nodal rhythm." The introduction of the electrocardiograph aided materially in solving this perplexing problem. Lewis and Rothberger and Winterberg almost simultaneously showed the relationship of experimental fibrillation of the auricles to characteristic electrocardiograms.

Various names have been given this arhythmia. Early observers, noting its frequent association with mitral disease, termed it the "mitral pulse." Hering described the arhythmia under the name of "pulsus irregularis perpetuus," and Gerhardt, "perpetual arhythmia."

Etiology.—In a recent analytic study of 500 cases of auricular fibrillation I obtained data as follows:

Both sexes are about equally affected, males 268 (53.6 per cent.) and females 232 (46.4 per cent.). The greatest age incidence occurred in the fifth and sixth decades (57.4 per cent.). Chronic endocardial valvular disease occurred in 153 cases (30.6 per cent.);

the lesions in order of frequency were (1) mitral regurgitation, 72 (46.1 per cent.); (2) double mitral lesion (regurgitation and stenosis), 40 (26.1 per cent.); (3) mitral stenosis, 28 (18.3 per cent.); and (4) aortic regurgitation, 13 (8.5 per cent.).

The greatest incidence of auricular fibrillation was with myocardial disease unassociated with endocardial valvular lesions, in 347 cases (69.4 per cent.). Myocardial disease grouped according to etiologic diseases in order of frequency were (1) hypertension with and without clinical nephritis, 98 (28.2 per cent.); (2) exophthalmic goiter, 97 (27.9 per cent.); (3) chronic myocarditis (true inflammatory disease), 81 (23.3 per cent.); and (4) adenoma with hyperthyroidism, 71 (20.5 per cent.). These data agree quite well with those of Levine published in 1917.

Three factors are probably responsible for the myocardial changes accompanying hypertension: (1) the cause or causes primarily responsible for the constitutional disorder; (2) the action on the myocardium of the retention products or of the intermediate products of incomplete metabolism or toxic agents resulting from imperfect renal or tissue function; and (3) the increased cardiac work, affecting largely the myocardium, resulting from the hypertension, *per se*, and the alterations in cardiovascular balance.

The myocardial degeneration secondary to hyperthyroidism is produced (1) by the cellular action of thyroxin (Kendall) on the myocardium, and (2) by the increased cardiac work accompanying the rise of the basal metabolic rate.

When auricular fibrillation supervenes in the course of heart disease it frequently remains as a permanent disorder. This is not always the case by any means, especially if the cause of the underlying heart disease can be removed with cardiac improvement, as in hyperthyroidism.

At times auricular fibrillation may be intermittent or transient. Occasionally paroxysmal fibrillation is encountered, behaving in

general like paroxysmal tachycardia, having a sudden inception, attended by a high rate, and terminating suddenly.

Experimental.—Auricular fibrillation is produced experimentally by mild faradization of the exposed animal auricle.

Lewis has recently shown, by very careful and exhaustive experimentation, the close relationship of the mechanism producing flutter and fibrillation. Both conditions have for their basis an abnormal area of stimulus production, namely, a circus movement following a central path in the auricle. Lewis groups the resulting disorders according to the degree of co-ordination of the abnormal mechanism. Pure flutter is that condition in which extraordinary regularity of auricular contractions occurs, and in which the excitation spreads uniformly over the auricle from cycle to cycle. Impure flutter is similar, but less regular, due to excitation dissemination in a less uniform manner. Fibrillation is considered as a more inco-ordinate status of the latter.

Mechanism.—The stimuli responsible for auricular fibrillation are extremely rapid, varying from 450 to 1000 a minute. The auricle no longer contracts, but its walls, dilated in diastole, are undergoing rapid, inco-ordinated fibrillary twitchings; it has lost its pump-like action and acts only as a reservoir in the general circulation.

Owing to the extremely rapid and inadequate production of stimuli only certain impulses are conducted through the auriculo-ventricular junctional tissues, resulting in disordered ventricular action.

When the ventricular rate is rapid many of the contractions are often ineffectual, that is, incapable of opening the aortic cusps, so that certain beats fail to reach the peripheral arteries. This results in a difference in the count between the apical pulse-rate and the radial pulse-rate and is termed the "pulse deficit." When the ventricular rate drops as the heart improves under rest and

treatment, the pulse deficit disappears and often proves to be a valuable index in the management of a patient.

The electrocardiographic findings in auricular fibrillation are very characteristic. A gross arhythmia which usually is total is at once apparent. In no two portions of the tracing are the cycle

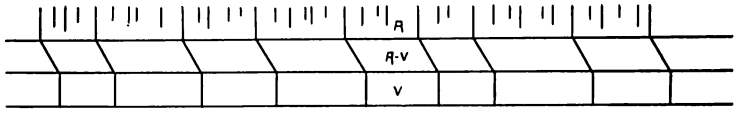

Fig. 40.—Schematic graph of coarse auricular fibrillation.

lengths uniform. Not infrequently variations in amplitude of the R waves occur. A most striking characteristic is the absence of the P wave. The reader is referred to Chapter III, in which the origin of the P wave is discussed. As the auricles no longer contract in fibrillation, the P wave is necessarily absent.

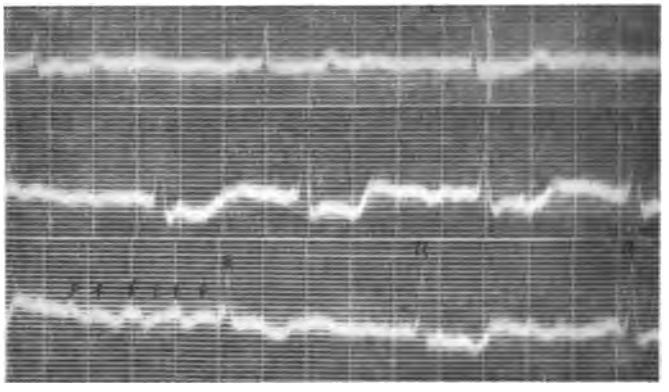

Fig. 41.—Coarse auricular fibrillation. Derivations I, II, and III. *ff*, Fibrillary.

Small irregular wavelets are often discernible, varying in rate, amplitude, and contour; they represent the fibrillary twitchings of the auricular musculature. They are denoted "f" in the electrocardiograms.

Hewlett and Wilson grouped their electrocardiograms of auricular fibrillation according to the character of the fibrillary wavelets

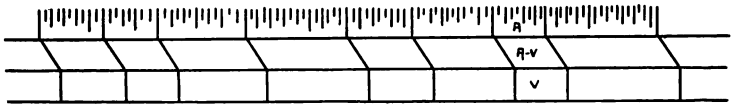

Fig. 42.—Schematic graph of fine auricular fibrillation.

into fine and coarse fibrillation. Figures 40 to 43 illustrate the two types. Figure 44 illustrates paroxysmal fibrillation.

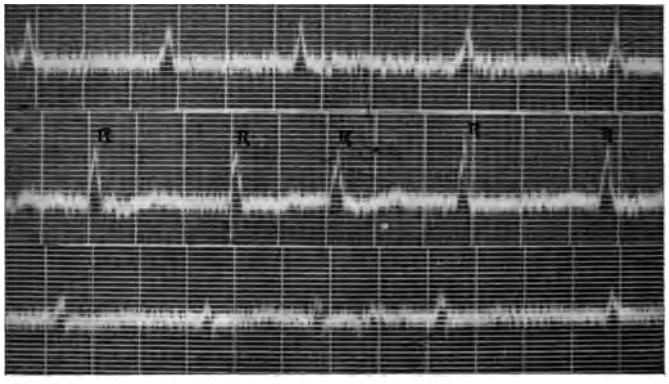

Fig. 43.—Fine auricular fibrillation. Electrocardiogram in Derivations I, II, and III.

Prognosis.—The presence of auricular fibrillation in varying degrees of heart disease renders a prognosis exceedingly difficult

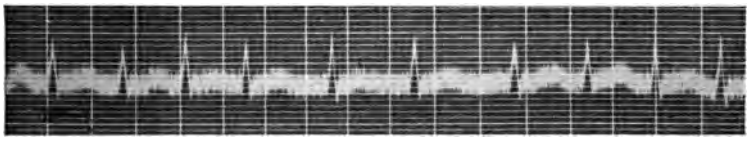

Fig. 44.—Paroxysmal auricular fibrillation. Rate 150. Electrocardiogram in Derivation II.

at times. Prognosis in heart disease, of course, cannot be based on one factor, but on the sum total of evidence, subjective and objective, and on knowledge gained by adjunct methods. Cardiac

efficiency is determined by the integrity of the myocardium, especially of the ventricular myocardium, as life is dependent directly on the function of these chambers.

Pardee believes that the prognosis of auricular fibrillation depends largely on proper treatment, and concludes "that the irregularity, *per se*, adds little or nothing to the gravity of the prognosis of the individual."

White studied the mortality in 100 cases of auricular fibrillation and compared the rate with that of 100 cases of normal rhythm. He found that 48 per cent. of the patients with auricular fibrillation and 47 per cent. of the patients with normal rhythm had died within three years.

In my study of 500 cases of auricular fibrillation during a period of four years I made a comparison of mortality figures with a control series which corresponded in total number, occurrence by decades and by sex, and represented as closely as possible similar disease conditions without fibrillation.

Group A. Complete series of auricular fibrillation (500 cases), cardiac mortality 41.3 per cent.

Group A. Control series (500 cases), cardiac mortality 13.6 per cent.

Group B. Uncomplicated auricular fibrillation (367 cases), cardiac mortality 36.9 per cent.

Group B. Control series, uncomplicated sinus rhythm (367 cases), cardiac mortality 16.2 per cent.

Group B_1. Auricular fibrillation with ventricular premature contractions (89 cases), cardiac mortality 42.0 per cent.

Group B_1. Control series, ventricular premature contractions (89 cases), cardiac mortality 15.9 per cent.

Group B_2. Auricular fibrillation with abnormal Q R S complexes in all derivations of the electrocardiogram (33 cases), cardiac mortality 63.4 per cent.

Group B_2. Control series, abnormal Q R S complexes in all derivations of the electrocardiogram (33 cases), cardiac mortality 54.2 per cent.

Group B_3. Auricular fibrillation with abnormal Q R S complexes in all derivations of the electrocardiogram and ventricular premature contractions (11 cases), cardiac mortality 87.5 per cent.

Group B_3. Control series, abnormal Q R S complexes in all derivations of the electrocardiogram with ventricular premature contractions (11 cases), cardiac mortality 87.5 per cent.

Group C. Auricular fibrillation in mitral regurgitation (72 cases), cardiac mortality 51.9 per cent.

Group C. Control series, mitral regurgitation (72 cases), cardiac mortality 27.8 per cent.

Group C_1. Auricular fibrillation in mitral stenosis (27 cases), cardiac mortality 47.1 per cent.

Group C_1. Control series, mitral stenosis (27 cases), cardiac mortality 22.2 per cent.

Group C_2. Auricular fibrillation in mitral regurgitation and stenosis (40 cases), cardiac mortality 53.4 per cent.

Group C_2. Control series, mitral regurgitation and stenosis (40 cases), cardiac mortality 34.6 per cent.

Group C_3. Auricular fibrillation in aortic regurgitation (13 cases), cardiac mortality 33.9 per cent.

Group C_3. Control series, aortic regurgitation (13 cases), cardiac mortality 71.4 per cent.

Group D. Auricular fibrillation in chronic myocarditis (81 cases), cardiac mortality 43.3 per cent.

Group D. Control series, chronic myocarditis (81 cases), cardiac mortality 43.3 per cent.

Group D_1. Auricular fibrillation in myocardial disease secondary to hypertension with or without clinical nephritis (98 cases), cardiac mortality 54.7 per cent.

Group D_1. Control series, myocardial disease secondary to hypertension with or without clinical nephritis (98 cases), cardiac mortality 53.8 per cent.

Group D_2. Auricular fibrillation in myocardial disease secondary to exophthalmic goiter (97 cases), cardiac mortality 34.2 per cent.

Group D_2. Control series, myocardial disease secondary to exophthalmic goiter (97 cases), cardiac mortality 17.1 per cent.

Group D_3. Auricular fibrillation in myocardial disease secondary to adenoma with hyperthyroidism (71 cases), cardiac mortality 30 per cent.

Group D_3. Control series, myocardial disease secondary to adenoma with hyperthyroidism (71 cases), cardiac mortality 8.9 per cent.

From these statistics it is apparent that the mortality attending auricular fibrillation doubles, and in some groups trebles, that occurring in similar types of heart disease not complicated by this arhythmia. The arhythmia, *per se*, is not the determining factor; the integrity of the myocardium plus proper treatment determines the patient's life expectancy.

VENTRICULAR FIBRILLATION

Ventricular fibrillation is analogous to auricular fibrillation. Records of ventricular fibrillation in man are extremely rare, as the condition is incompatible with life. Robinson and Bredeck, however, have reported a case in which there was temporary recovery from this abnormal ventricular mechanism. Ventricular fibrillation is probably the terminal disorder in many deaths. Records obtained at the time of death will solve this speculation.

Experimental.—Mild faradization of the ventricles induces fibrillation. Lewis has shown that obstruction of the coronary vessels may lead to fibrillation of the ventricles. Levy demonstrated that light chloroform anesthesia predisposes to ventricular

fibrillation, and later he produced this disordered cardiac action in dogs by the injection of epinephrin in conjunction with light chloroform anesthesia.

Mechanism.—The mechanism of ventricular fibrillation is identical with that of fibrillation of the upper chambers. The

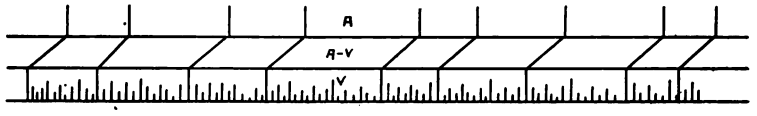

Fig. 45.—Schematic graph of ventricular fibrillation.

fact that the ventricles no longer contract makes the condition incompatible with life if it is maintained for more than a very transient period. Figure 45 illustrates the mechanism. Figure 46 is an electrocardiogram taken in Derivation II of a goat just preceding and during death. The auricular complex P and the con-

Fig. 46.—Paroxysm of ventricular fibrillation just preceding death. Electrocardiogram of a goat in Derivation II.

traction wave T of the ventricles are absent. The ventricular complexes Q R S are rapid, inco-ordinate, and bizarre.

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CHAPTER VI

ECTOPIC RHYTHMS AND TACHYCARDIAS

WHEN the cardiac impulse arises at a point outside the sino-auricular node (pace-maker) the resulting rhythm is referred to as ectopic. The ectopic focus may be in the auricle, in the ventricle, or in the junctional tissues. Ectopic rhythms have been classified by Lewis[16] into two groups, the homogenetic and the heterogenetic.

The first type is characterized by a gradual onset, the rate is relatively slow, the shift of the pace-maker may be gradual or abrupt, and the seat of ectopic impulse production is probably always within the system of specialized tissue (conduction system). The heart is under the control of its extrinsic innervation. This type probably results from exaggerated physiologic processes.

The second type, the heterogenetic, has a sudden onset and rapid rate, and the location of the pace-maker is abruptly shifted. The abnormal pace-maker may be within the specialized tissue or without. The heart is not under the control of its extrinsic innervation. This type is believed to result from pathologic processes.

The various types of paroxysmal tachycardia, with the exception of paroxysmal sinus tachycardia, are ectopic rhythms. To prevent confusion paroxysmal sinus tachycardia will also be considered in this chapter.

PAROXYSMAL SINUS TACHYCARDIA

The only characteristic feature of paroxysmal sinus tachycardia is the sudden inception of rapid rate. Impulse origin in the normal pace-maker gives an electrocardiogram which does not materially

deviate from the normal. When the rate is extremely rapid the T wave loses its identity by submersion, but proof of its presence is found by measuring the interval between the last normal com-

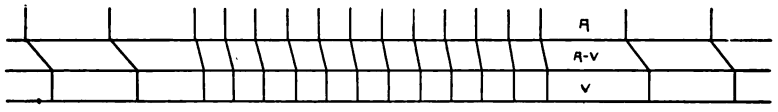

Fig. 47.—Schematic graph of paroxysmal sinus tachycardia.

plex preceding the paroxysm and the first normal complex after the attack, showing that retrogression has not occurred. The individual waves of these electrocardiograms are unaltered.

Fig. 48.—Paroxysmal sinus tachycardia. Rate 55 to 136. Electrocardiogram in Derivation II.

Figure 47 represents this disorder and Fig. 48, the termination of a paroxysm.

PAROXYSMAL AURICULAR TACHYCARDIA

In auricular tachycardia the ectopic focus of stimulus production is at some point in the auricular musculature. Each auricular

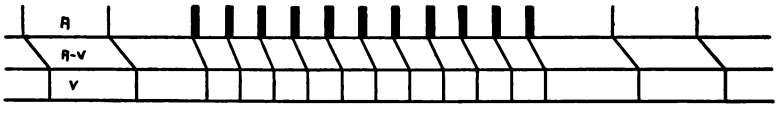

Fig. 49.—Schematic graph of paroxysmal auricular tachycardia.

beat is followed by a ventricular beat, and the rate does not attain 200 a minute. Figure 49 schematically represents a paroxysm

of auricular tachycardia. Figure 50 is an electrocardiogram of a paroxysm. It is apparent that 1 : 1 auriculoventricular association occurs. The P wave is inverted, which is indicative of auricular impulse production outside the sino-auricular node.

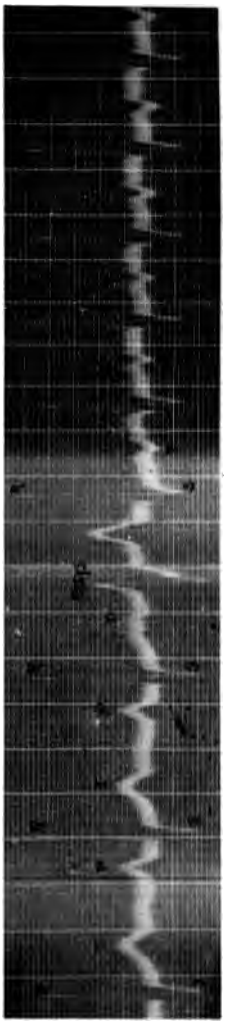

Fig. 50.—Onset of a paroxysm of auricular tachycardia. Sinus rate 86. Tachycardia rate 150. Paroxysm lasted twenty-three and one-half seconds. Electrocardiogram in Derivation II.

AURICULAR FLUTTER

Auricular flutter may be described as an acceleration of the auricles to a rate beyond 200 a minute. This acceleration is accompanied by partial heart-block, giving a ventricular rate of one-half, one-third, or one-fourth of the auricular rate, or a complete auriculoventricular dissociation (complete heart-block), or varying degrees of partial block, causing a gross ventricular arhythmia. The partial block is apparently due to the inability of the auriculoventricular bundle to conduct impulses so rapidly, or to the inability of the ventricle to respond so rapidly.

There is no known pathologic difference between an auricular rate of less than 200 and one exceeding this figure, yet the clinical manifestations are so different as to justify the classification of flutter as a clinical entity. The fundamental clinical differences lie in the fact that flutter tends to persist indefinitely, whereas auricular paroxysmal tachycardia rarely reaches so rapid a rate,

and the attack stops after a relatively short period. In flutter the auricles continue their rapid rate when the ventricles are slower, while in auricular paroxysmal tachycardia 1 : 1 rhythm is always present and the sinus rhythm is restored between attacks.

Etiology.—The following data are based on a study of 16 cases[1] of auricular flutter in the Mayo Clinic and of 59 cases reported in the literature: The disorder occurred four times as often in men as in women. The average age of the patients was forty-seven years, the youngest six years, and the oldest eighty-two years. The condition was most frequent between the ages of forty and sixty years in the combined series, but in our series more cases occurred between thirty and forty years.

Our cases call attention to an etiology of infection, since antecedent diseases of probable streptococcic origin were noted with remarkable frequency, namely, rheumatic fever 1 case, tonsillitis 5 cases, dental sepsis 6 cases, "grippe" 6 cases, and pneumonia 2 cases. In three instances the patient dated his symptoms from one of these infections. All the patients in our series gave histories of one or more of the foregoing diseases. In the cases in the literature the etiology was usually not given, but the streptococcus group predominates; thirteen histories of rheumatic fever are recorded.[4, 12, 24, 27, 30, 31, 33, 34] Venereal disease plays no evident part. None of our patients had syphilis, although 3 cases are noted in the literature.[2, 4, 31]

Exophthalmic goiter was definite in 4 of our cases and was believed to be the probable etiologic factor. One such case is reported in the literature.[31] Mitral disease was observed in but 1 of our 16 patients; in the literature are reports of 10 cases of stenotic or double mitral lesions.[15, 22, 29, 31]

Experimental.—Auricular flutter was produced experimentally by MacWilliam, in 1887, by mild faradization of the auricles of exposed animal hearts. Lewis[14] observed the same condition

after the intravenous injection of glyoxylic acid. Hirschfelder produced it by ligation of the coronary arteries, and similar observations have been made after cooling the auricles and during chloroform anesthesia. In our work with Kendall in the laboratories at the Mayo Clinic we produced experimental hyperthyroidism in the goat by large intravenous injections of thyroxin. Auricular flutter was one of the disorders of cardiac action observed during continuous electrocardiographic tracings during several hours preceding death.

Mechanism.—Flutter is caused by a focus of stimuli in the wall of the auricular muscle at a point outside the normal pacemaker or sinus node (ectopic stimuli), the discharge of stimuli being at a rate so rapid and continuous as to submerge the sinus activity. This conclusion is based on the fact that the P wave is found to be inverted and to have an increased amplitude.

Lewis has recently very clearly depicted the true nature of flutter. The impulses resulting from the abnormal area of stimulus production are diffused by a circus movement following a central path in the auricle. Lewis emphasizes the remarkable regularity of clinical flutter, showing by careful measurements that the variations in the lengths of intra-auricular cycles average less than 0.0009 to 0.0077 second in curves of from fourteen to thirty-two auricular cycles. Tracings showing less regularity are termed "impure flutter" and result from less uniform impulse diffusion. This status is intermediate between pure flutter and fibrillation.

In the human heart the irritable focus causing flutter must be the result of disease. It cannot be emphasized too strongly that flutter, *per se*, is only objective evidence of localized irritability in the auricular wall, and that any other organic cardiac disease may exist in the same heart.

It is evident that auricular flutter is not a pathologic entity, for we often see auricular premature contractions, flutter, fibrilla-

tion, and a sinus rhythm in the same patient within a relatively short time. The literature contains only 6 necropsy reports in unquestioned cases; our series contains 2 others. Ritchie[30] has reported a lymphocytic infiltration of the epicardium, most marked in the region of the sinus node, and he thinks this may have depressed sinus activity. The case of Mackenzie and Humes' first case add nothing significant to these findings.

The irritable auricular focus is the essential feature, and our study must include all causes of localized injury or irritability to heart muscle. Such causes may be classified under three heads: (1) infections causing localized injury; (2) general and local myocardial degeneration from any cause, such as hypertension, valvular disease, and hyperthyroidism; and (3) localized malnutrition of the auricular wall, as in vascular sclerosis, and so forth. We do not know why in certain cases a localized injury should be selected from more extensive myocardial damage to become a source of irritation, and to send forth such rapid impulses as to submerge the sinus rate and establish flutter. That such functional pathology exists, however, is evident. We have no evidence that flutter can be purely of neurogenic origin.

The electrocardiograms of flutter are characterized by the extremely rapid auricular rate, more than 200 per minute, and by inversion of the auricular P wave. Partial heart-block is usually present, the auriculoventricular ratio varying from 2 : 1 to 5 : 1; occasionally paroxysm of 1 : 1 association occurs, and again, complete heart-block may be present.

Figure 51 illustrates the mechanism in flutter. Figure 52 shows a paroxysm of 1 : 1 flutter; Fig. 53, 2 : 1 flutter; Fig. 54, 3 : 1 flutter; Fig. 55, 4 : 1 flutter; Fig. 56, 5 : 1 flutter; Fig. 57 shows flutter with complete heart-block.

Flutter may be conveniently classified as paroxysmal or chronic, depending on the duration of the disorder. We use the term

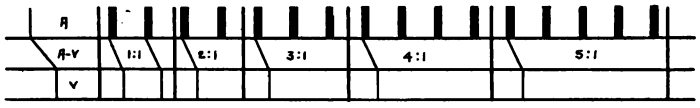

Fig. 51.—Schematic graph of auricular flutter.

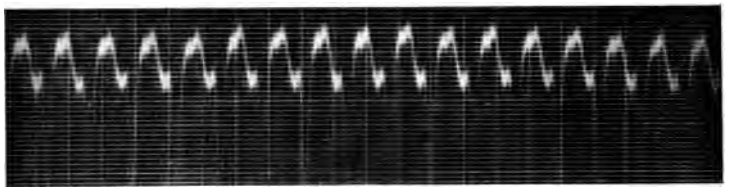

Fig. 52.—Auricular flutter, 1 : 1 rhythm. Rate 232. Electrocardiogram in Derivation II.

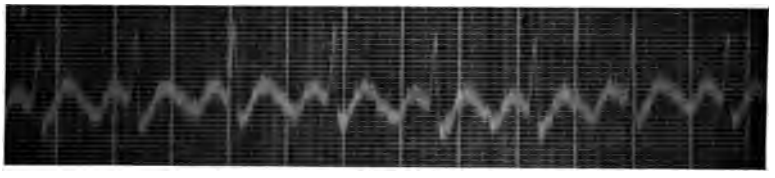

Fig. 53.—Auricular flutter, 2 : 1 rhythm. Auricular rate 324; ventricular rate 162. Electrocardiogram in Derivation II.

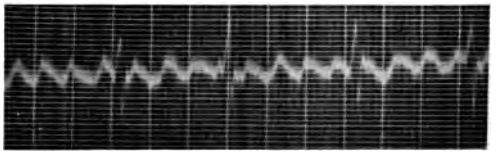

Fig. 54.—Auricular flutter, 3 : 1 rhythm. Auricular rate 270; ventricular rate 90. Electrocardiogram in Derivation II.

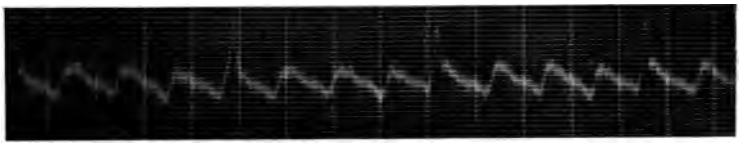

Fig. 55.—Auricular flutter, 4 : 1 rhythm. Auricular rate 240; ventricular rate 60. Electrocardiogram in Derivation II.

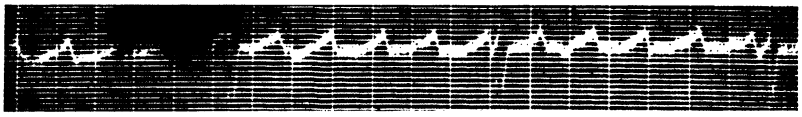

Fig. 56.—Auricular flutter, 5 : 1 rhythm. Auricular rate 210; ventricular rate 42. Electrocardiogram in Derivation III.

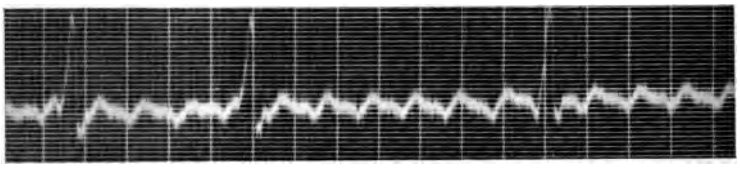

Fig. 57.—Auricular flutter with complete heart-block. Auricular rate 260; ventricular rate 43. Electrocardiogram in Derivation II.

"paroxysmal" in cases in which the normal rhythm is restored between attacks lasting a few hours or days, and "chronic" when the condition tends to persist.

Paroxysmal flutter is not clearly defined from auricular paroxysmal tachycardia, as previously mentioned. It has been observed in the Clinic only as a disorder incidental to evident myocardial disease; it is serious because of the great strain on the myocardium.

Chronic flutter should always be recognized, for usually it can be relieved. The flutter lasts for long periods, for weeks or even years, and can be detected by proper tracings at any time during its course. The ordinary auriculoventricular ratio is 2 : 1, and the pulse is usually from 100 to 180, but any degree of block may exist.

It is not my intention to consider therapeutics in this work, but the treatment of flutter is so important as to merit mention in this connection. Lewis[21] has shown that digitalis is the sovereign remedy in flutter. Digitalis is administered until fibrillation supervenes, when the drug is discontinued and frequently normal sinus rhythm is restored. At times, however, the stage of fibrillation is followed by flutter, when treatment must be re-established.

We have observed no ill effects from massive digitalis dosage except the temporary toxic symptoms, and we feel sure that most patients require massive dosage to obtain the desired result. Furthermore, we have found better results from pushing the drug to physiologic complete block if the patient tolerates it to this

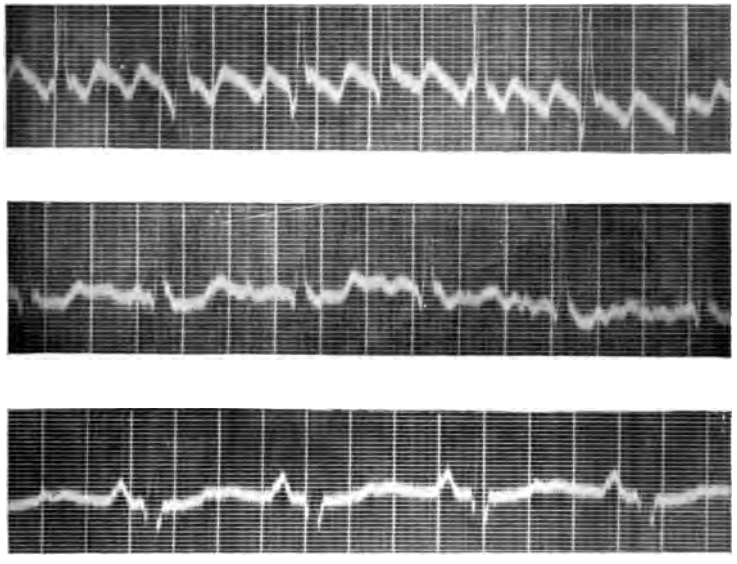

Fig. 58.—The effect of digitalis in auricular flutter. Derivation I, auricular flutter, 2 : 1 rhythm. Derivation II, auricular fibrillation, twelve days later. Derivation III, sinus rhythm, thirty-three days later. Electrocardiograms in Derivation II.

point, that is, far beyond the point of fibrillation in most instances. Figure 58 illustrates the effect of treatment.

AURICULOVENTRICULAR (NODAL) RHYTHM

That the auriculoventricular node is endowed with the inherent property of automatism was first recognized by Engelmann[3] following his application of the first Stannius ligature in the frog. Ordinarily this property of the auriculoventricular node is not apparent because of the greater rhythmicity of the sinus node. The frequent

and regular stimuli which issue from the normal pace-maker render the stimuli of the auriculoventricular node subminimal. The rate of the latent inherent rhythm of the auriculoventricular node has been termed its "period" by Williams and James,[36] and may be approximately that of the sinus rate or less.

Experimental.—Auriculoventricular rhythm has been produced experimentally by two methods: (1) depressing sinus activity to the point where the auriculoventricular node by virtue of its inherent automatism asserts itself and dominates the cardiac rhythm, and (2) increasing the irritability of the auriculoventricular node, thereby exalting its automatism.

Depression of sinus activity has been accomplished (1) by cooling the sinus node with an ice pencil or by the use of ethyl chlorid, (2) by crushing or excising the sinus node, and (3) by isolating the node by making encircling incisions. Excitation of the auriculoventricular node has been accomplished in various ways. Lewis[17] and Meek and Eyster were able to produce auriculoventricular rhythm by stimulation of the right vagus. By stimulation of the left accelerator Rothberger and Winterberg obtained the same effect.

Clinically, two varieties of auriculoventricular rhythm occur: (1) the homogenetic type, characterized by a relatively slow rate, with a gradual onset and termination, and (2) the heterogenetic or true paroxysmal tachycardia type, with a rapid rate, sudden inception, and sudden termination. The homogenetic type has been produced in man in various ways. Wilson[37, 38] has produced it by forced respiration and by the hypodermic injection of atropin. The heterogenetic type is a relatively rare condition.

Graphically, three types of auriculoventricular rhythm are recognized, depending on the site of origin in the node. Hering has shown that with normal rhythm most of the delay between auricular and ventricular systoles occurs in the auriculoventricular

node. In line with this is the observation that the P–R interval of the electrocardiogram is diminished in auriculoventricular rhythm, and that this diminution bears a relationship to the node level at which the ectopic rhythm originates.

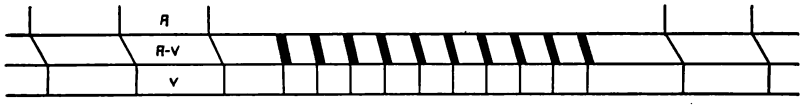

Fig. 59.—Schematic graph of paroxysmal nodal tachycardia showing diminished P–R interval.

1. The P–R interval is reduced when the rhythm originates in the upper portion of the node. The schematic graph (Fig. 59) portrays this, as does the electrocardiogram (Fig. 60).

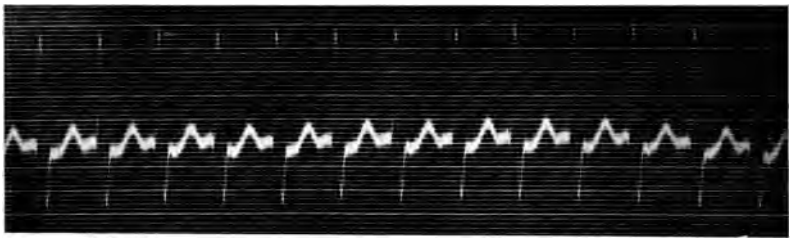

Fig. 60.—Paroxysmal nodal tachycardia. Diminished P–R interval. Rate 164. Electrocardiogram in Derivation II.

2. The P–R interval is absent (0) when the rhythm originates in the lower portion of the node. Figures 61 and 62 represent this type.

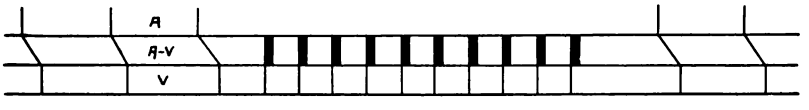

Fig. 61.—Schematic graph of paroxysmal nodal tachycardia. P–R interval 0.

3. The R–P interval is present when the rhythm originates in the lowest portion of the node or of the bundle (Figs. 63, 64). Meakins has called attention to the fact that the P wave is frequently inverted in auriculoventricular rhythm.

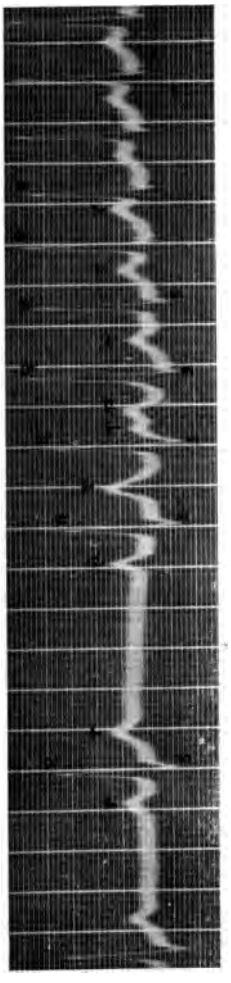

Fig. 62.—Onset of a paroxysm of nodal tachycardia. P–R interval 0. Sinus rate 50. Tachycardia rate 150. Electrocardiogram in Derivation II.

Fig. 63.—Schematic graph of paroxysmal nodal tachycardia showing R–P interval.

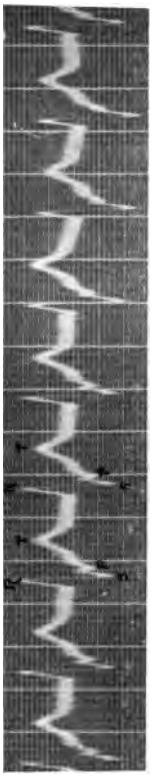

Fig. 64.—Paroxysmal nodal tachycardia. R–P interval. Note the inversion of P wave.

VENTRICULAR TACHYCARDIA

Ventricular tachycardia is a rare disorder of the cardiac mechanism. It is of the heterogenetic type.

Etiology.—The rarity of the condition affords little data concerning the etiology. I have been afforded the opportunity of studying 5 patients with ventricular tachycardia; 3 were males

and 2 were females; the youngest was twenty-one years, the oldest sixty-two years; the average age was forty-one years. Four of the patients gave definite histories of previous infection with the streptococcus group. Syphilis was not determined in any case. In one fatal case marked atheroma of the left coronary artery was found.

Experimental.—Regular series of suitably arranged induction shocks produce series of premature ventricular contractions simulating the graphic records of ventricular tachycardia. Lewis[13] constantly produced premature ventricular contractions by ligating the coronary arteries by tying off the left descending branch, and in most instances by impairing the circulation in the right vessel. As the nutrition of the ventricle became progressively impaired, series of heterogenetic contractions occurred, the sequence becoming longer as the nutritional changes became more marked.

Rothberger and Winterberg produced ventricular tachycardia by the intravenous injection of salts in dogs. They found that combined stimulation of the vagi and accelerators caused cessation of the heart-beat, but after the injection of from 5 to 10 mg. of barium chlorid in 1 per cent. aqueous solution premature ventricular contractions occurred. With doses of 25 to 50 mg., minus accelerator stimulation, ventricular tachycardia, and, at times, a transient arhythmia were produced. Calcium chlorid, 100 to 200 mg. in 10 per cent. aqueous solution, produced similar results. Rothberger and Winterberg concluded that these salts increase the ventricular irritability, but they did not believe that the nodal tissues were appreciably affected.

The electrocardiogram exhibits series of premature ventricular contractions, the complex forms varying with the point of origin in the ventricles. Identification of auricular contractions during the tachycardia is frequently difficult, but careful measurement shows that retrogression does not occur, since the first auricular

complex of the normal rhythm falls at the proper point (Figs. 65 to 68).

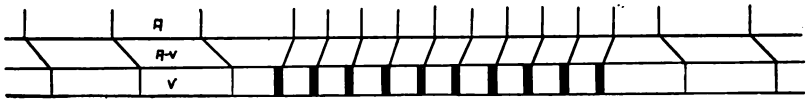

Fig. 65.—Schematic graph of paroxysmal ventricular tachycardia.

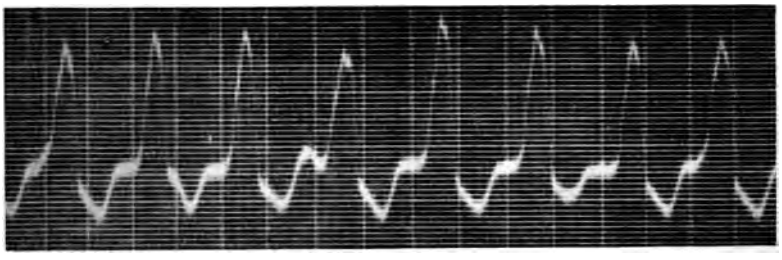

Fig. 66.—Paroxysm of ventricular tachycardia. Rate 133. Electrocardiogram in Derivation II.

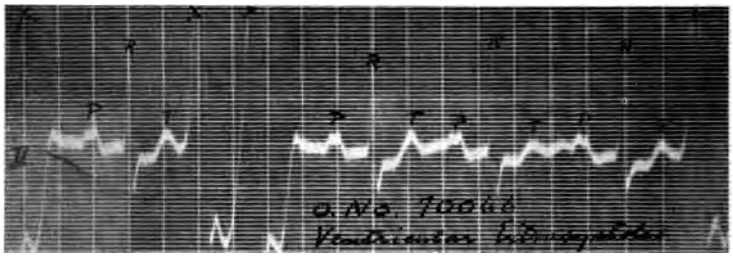

Fig. 67.—Premature ventricular contractions preceding a paroxysm of ventricular tachycardia. Electrocardiogram in Derivation II.

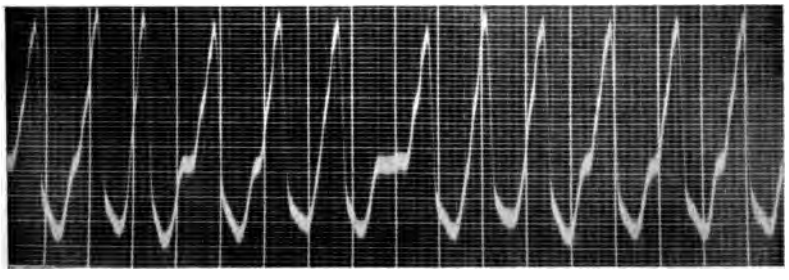

Fig. 68.—Paroxysm of ventricular tachycardia. Rate 180. Electrocardiogram in Derivation I.

The auricles and ventricles contract at the same rate, for each complex is identical to the adjacent complex, and if auricles and ventricles were contracting at independent rates, the auricular complex would at times be superimposed and destroy the contour of the general curve. Rarely does the rate of the ventricles exceed that of the auricles, as in Palfrey's case.

In all probability, any condition increasing ventricular irritability is a potential factor in the production of ventricular tachycardia, and until more necropsy material is available conclusions with regard to types of lesions must remain hypothetic. The gravity of the condition depends, of course, on the degree of myocardial damage and the duration of the paroxysms. The maintenance of circulation is dependent on ventricular action and not on auricular action, and obviously this abnormal ventricular rhythm must be regarded as potentially a grave disorder. Lewis emphasized this point, stating that ventricular tachycardia borders on fibrillation; ventricular fibrillation, so far as we know, is incompatible with life.

VENTRICULAR ESCAPE

Occasionally the ventricles escape from the control of the sino-auricular node through the independent autonomy of the auriculo-ventricular node.

White has called attention to two types of ventricular escape, that which results from depression of the pace-maker in the sino-auricular node, and that which results from excitation of the pace-maker in the auriculoventricular node. The latter is extremely rare. The identification of the two types rests largely on the rate of the sino-auricular pace-maker from which the ventricles escape, and on the rate of the ventricular pace-maker which escapes.

The distinction between ventricular escape and auriculoventricular rhythm depends entirely on the electrocardiogram. In

ventricular escape the heart is under control of two pace-makers, while in auriculoventricular rhythm only the junctional node

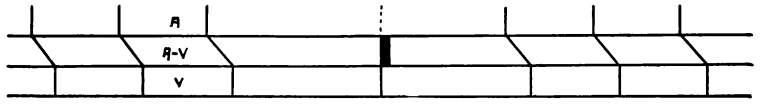

Fig. 69.—Schematic graph of ventricular escape.

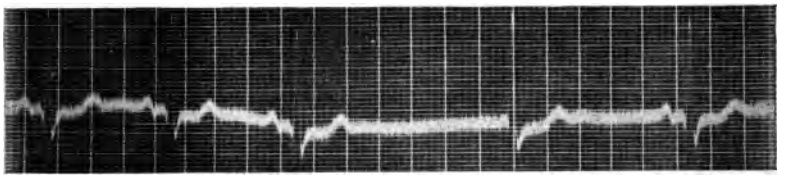

Fig. 70.—Ventricular escape. Electrocardiogram in Derivation II.

functionates. Occasionally the ventricular rate exceeds that of the auricles, as in 3 of White's cases (Figs. 69, 70).

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CHAPTER VII

DISORDERS OF CARDIAC CONDUCTION

IMPULSE transmission along the normal conduction paths of the heart may be interrupted by the invasion of disease or by the augmentation of the vagus mechanism.

COMPLETE AURICULOVENTRICULAR DISSOCIATION (COMPLETE HEART-BLOCK)

Gaskell, in 1883, working on the hearts of the tortoise and of the frog, demonstrated muscular conduction from auricle to ventricle. By clamping the auriculoventricular ring the auricle continued to beat with an unaltered rhythm, but as the clamp was tightened the periods between auricular and ventricular contractions were gradually lengthened. Gradually the ventricle failed to respond to certain auricular contractions or failed to respond at all. When the clamp was closed very tightly ventricular standstill occurred for a variable period when its inherent rhythm asserted itself. Gaskell further showed that heart-block resulted when the muscular band between the auricles and ventricles was cut.

Mechanism.—Complete auriculoventricular dissociation is due to failure of the auricular impulse to pass along the auriculoventricular bundle. Thus the ventricle is not excited to contraction by the impulses arising in the sino-auricular node, but assumes an independent rhythm which is slow and at times arhythmic.

Etiology.—Permanent complete auriculoventricular dissociation is associated with disease of the auriculoventricular bundle obstructing impulse conduction, although Hume, Krumbhaar, and Pepper and Austin have reported cases in which no lesion was demonstrable. Cases of transient complete heart-block have been reported by Cohn, Cohn, Holmes, and Lewis, Heard and

Colwell, and Wilson and Robinson, and our records reveal 2 cases, evidently due to vagus augmentation.

Some of the lesions that have been found to obstruct the auriculoventricular bundle have been collected from the literature and tabulated by Hirschfelder as follows: gumma, 7; calcified patches involving the bundle, 4; fibrosis of the bundle, 6; tumors in the septum, fibroma 1 and round-cell sarcoma 1; anemic infarction of the bundle, 2; simple round-cell infiltration of the bundle, 1; mural ulceration involving the bundle (ulcerative endocarditis), 1; fatty degeneration, 1; arteriosclerosis of the artery supplying the bundle, 1.

In an analysis of 22 cases of complete auriculoventricular dissociation the apparent etiologic conditions were chronic myocarditis 50 per cent., chronic endocardial valvular disease 22.7 per cent., myocardial degeneration associated with the hypertension group 22.7 per cent., and cardiosclerosis 4.5 per cent. Syphilis was not demonstrated in a single instance.

The electrocardiograms of complete auriculoventricular dissociation are characteristic. One is impressed with the slow ventricular rate, usually in the neighborhood of 30 each minute. The Q R S complex is often bizarre, the limbs or apex being notched. At times a gross ventricular arhythmia is present. The auricular rate is usually relatively rapid; the P waves occurring at regular

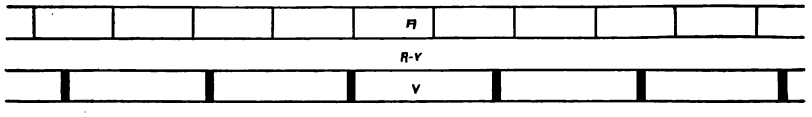

Fig. 71.—Schematic graph of complete auriculoventricular dissociation.

intervals bear no relationship to the ventricular complexes. At times the P wave is superimposed on the R wave or the T wave, deforming the respective wave during that cycle. Occasionally the P wave is notched (Figs. 71 to 74).

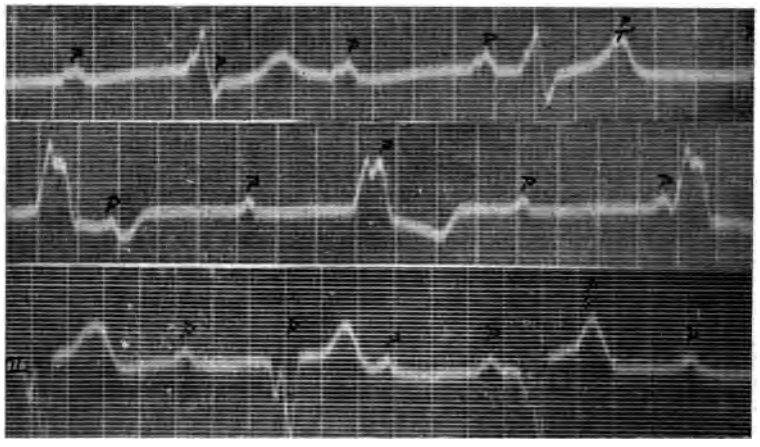

Fig. 72.—Complete auriculoventricular dissociation; ventricular rate 35; auricular rate 82. Electrocardiogram in Derivations I, II, and III.

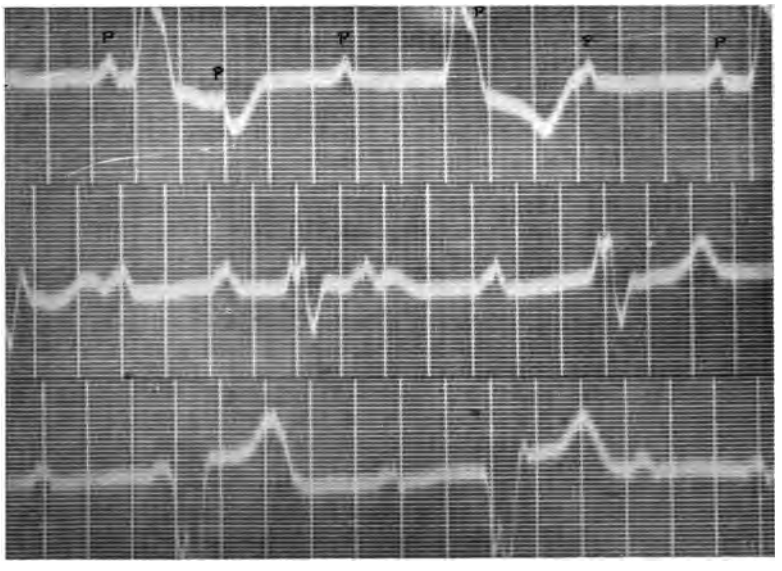

Fig. 73.—Complete auriculoventricular dissociation; ventricular rate 40; auricular rate 100. Electrocardiogram in Derivations I, II, and III.

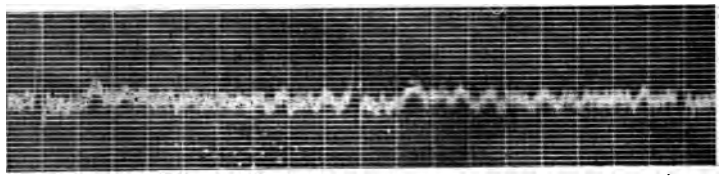

Fig. 74.—Complete auriculoventricular dissociation with auricular fibrillation. Rate 33. Electrocardiogram in Derivation II.

The Adams-Stokes' seizures so frequently occurring in complete auriculoventricular dissociation are due to cerebral anemia resulting from ventricular standstill, or from diminution, to a marked degree, of volume outflow of blood. Mackenzie's observations have shown that ventricular standstill for ten seconds produces unconsciousness, and for twenty seconds, general convulsions of the body.

PARTIAL HEART-BLOCK

Partial heart-block consists in the absence of ventricular response to certain auricular impulses. It may result from such rapid auricular contraction that the conductivity of the auriculoventricular bundle permits only certain impulses to reach the ventricles, as in auricular flutter. The irritability of the bundle may be reduced so that only certain impulses are conveyed to the

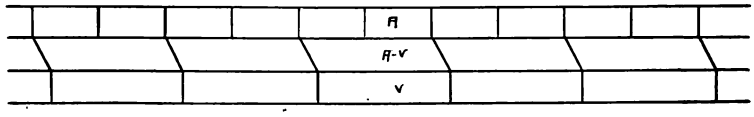

Fig. 75.—Schematic graph of partial block.

ventricle. The irritability of the ventricles may be diminished so that they fail to respond to all the impulses that reach them.

A geometric ratio exists between auricular and ventricular beats so that the ventricle responds to every second, third, fourth, or fifth impulse. The ventricular rhythm is regular unless the

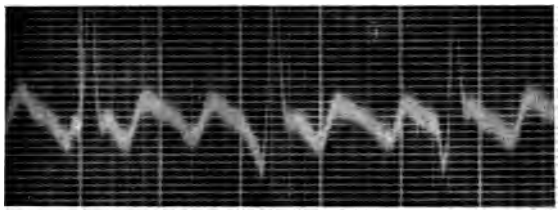

Fig. 76.—Partial block, 2 : 1 rhythm. Electrocardiogram in Derivation II.

Fig. 77.—Varying degree of partial block, 2 : 1 and 4 : 1 rhythm. Electrocardiogram in Derivation III.

degree of partial block varies from cycle to cycle, when a gross irregularity results. The irregularity at times is total and clinically cannot be distinguished from auricular fibrillation (Figs. 75 to 77).

DELAYED AURICULOVENTRICULAR CONDUCTION

The normal conduction time between the auricles and the ventricles (P–R interval) does not exceed 0.22 second. Many observers consider 0.20 second the upper limit of normal, although I believe that 0.22 second conduction time not infrequently is observed in normal hearts.

Delay in impulse transmission through the bundle may be the result of injury by disease, or by depression of its activity by vagus stimulation. Digitalis given to full physiologic effect also delays conduction by its action on the vagus.

In the electrocardiogram the P–R interval shows prolongation beyond 0.22 second. Neuhof published an electrocardiogram

showing extreme prolongation, 0.80 second. In order to determine whether or not the prolonged P–R interval is the result of organic bundle changes or of vagus augmentation tracings should be taken before and after the administration of atropin. At times the rate is slow, 50 to 60 each minute, but not always, by any means, since tachycardia is sometimes found (Figs. 78 to 80).

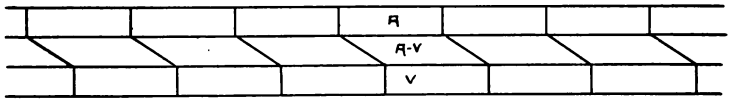

Fig. 78.—Schematic graph of delayed auriculoventricular conduction.

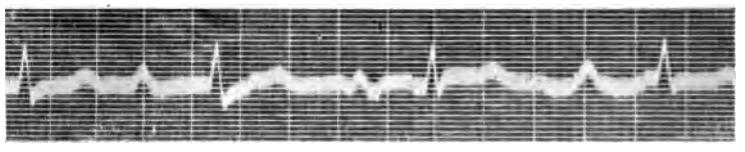

Fig. 79.—Delayed auriculoventricular conduction. P–R interval 0.31 second. Electrocardiogram in Derivation II.

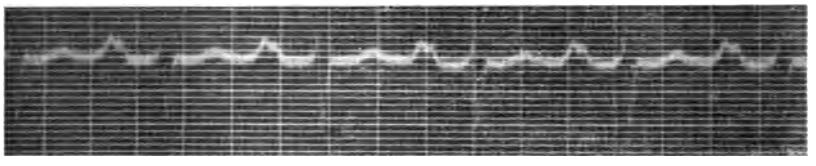

Fig. 80.—Delayed auriculoventricular conduction. P–R interval 0.26 second. Electrocardiogram in Derivation I.

SINO-AURICULAR HEART-BLOCK

Sino-auricular heart-block, or transient cardiac standstill; is a rare disorder of the cardiac mechanism. Occasionally abnormal influences affect the cardiac vagus, disturbing the function of the sino-auricular node so that an entire beat is blocked. Such cases have been reported by Eyster and Evans, Levine, Eyster and Meek, and Brown.

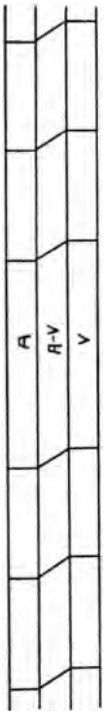

Fig. 81.—Schematic graph of sino-auricular block.

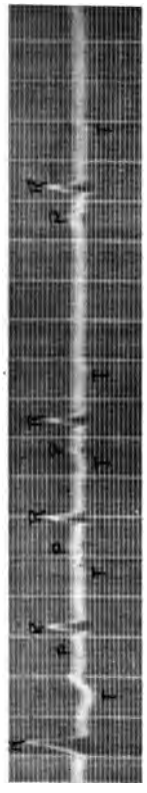

Fig. 82.—Sino-auricular block. Electrocardiogram in Derivation I.

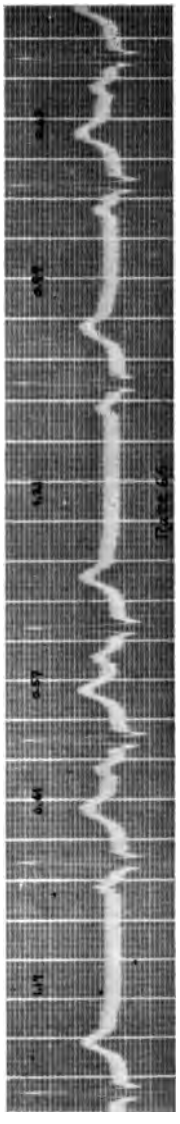

Fig. 83.—Sino-auricular block. Electrocardiogram in Derivation II.

Hewlett, White, and Parkinson have observed sino-auricular heart-block during the administration of digitalis. Sicard and Meara have observed it from salicylic acid; Cushny, from aconitin;

Cohn, after morphin administration; and Neuhof, in tabagism. These various substances described as producing sino-auricular heart-block probably did so by augmenting vagus action.

The failure of the auricle to contract may result from (1) failure or weakness of impulse production, (2) muscle weakness, and (3) blocking the impulse between the sino-auricular node and the auricles. The electrocardiogram reveals standstill of the entire heart, the pause between complexes during the block being usually somewhat less than the interval between two normal cycles. In extreme instances the pause may equal the time required for three or even four normal cycles (Figs. 81–83).

ABERRANT Q R S COMPLEXES IN ALL DERIVATIONS

Much interest has been displayed the last few years in changes affecting the Q R S complex of the electrocardiogram. Numerous interpretations have been given these changes. The changes constituting an abnormal Q R S complex are a base width exceeding 0.10 second in complexes of unaltered contour, and in aberrant complexes, notching or splintering of the ascending or descending limb or of the apex R. The complexes may be of low or high amplitude and monophasic or diphasic.

Complexes of High Amplitude.—Eppinger and Rothberger were the first to associate changes in the Q R S complex with lesions of the two main branches of the auriculoventricular bundle. When the right branch was cut, the normal ventricular complex was replaced by a diphasic complex of high amplitude. The Q R S complex was directed downward, the base width was prolonged, and the T wave was directed upward and exaggerated in a derivation from the esophagus and the rectum. Division of the left branch revealed similar changes except that reversal in direction of the complexes occurred.

In 1910 Eppinger and Stoerk published the results of an electro-

cardiographic study dealing with bundle branch block. Two of their patients came to necropsy. In both cases lesions were demonstrated completely dividing the right branch. The electrocardiograms showed diphasic Q R S complexes in all derivations of increased amplitude in Derivations I and III and having a base width exceeding the normal. The Q R S complex was directed upward in Derivation I and downward in Derivations II and III.

In 1913 Rothberger and Winterberg investigated the subject, using the usual three derivations. After division of one of the main bundles they obtained typical diphasic complexes, but the Q R S complex in Derivations I and III had the same and not the opposite direction, as in the patients reported by Eppinger and Stoerk.

Lewis (1916), prompted by the variance of views regarding bundle branch block, conducted a series of experiments on dogs. After division of the right branch of the bundle in most instances the Q R S complex was directed downward in all derivations; these electrocardiograms were called "concordant." In a few animals the Q R S complex was directed upward in Derivation I; these tracings were termed "discordant." The fact that right bundle branch block in man produced discordant electrocardiograms, and in most dogs produced concordant curves, was an apparent inconsistency, but Lewis showed that the discrepancy resulted from anatomic differences in the hearts. The hearts which produced discordant curves showed less bridging of the cavity of the left ventricle by ramifications of the left branch than the hearts which produced concordant curves.

Recently Fahr, from purely theoretic considerations, stated that the usual diagnosis of right and left bundle branch lesions is probably wrong. He believes that what has been interpreted as a right bundle block is, in reality, a block of the left branch. The experimental work cited, however, certainly has greater reliability than pure hypothesis.

Carter, in 1914, published electrocardiograms as examples of bundle branch block and tabulated data differentiating the normal and aberrant complex as follows:

CONTRASTS BETWEEN NORMAL AND ABERRANT ELECTROCARDIOGRAMS

NORMAL	ABERRANT
1. Supraventricular complex. Presence of auricular or P summits.	1. Supraventricular complex. Presence of auricular or P summits.
2. The P–R interval 0.13 to 0.18 second, never more than 0.2 second.	2. P–R interval frequently prolonged beyond 0.2 second.
3. Q R S interval less than 0.1 second and less than one-third of entire complex.	3. Q R S interval exceeds 0.1 second and as a rule constitutes more than one-third of entire complex.
4. Relatively small amplitude of initial deflections.	4. Relatively increased amplitude of initial deflections.
5. Final deflection T upright and in the same direction as the most prominent deflection (R) in Derivations I and II, and usually in Derivation III.	5. Final deflection T′ usually in a direction opposite to that of the prominent initial deflection.
6. Initial deflections, as a rule, unnotched.	6. Initial deflections almost always show notching in one derivation at least. Many bizarre forms seen.
7. Final deflection T, as a rule, plainly to be seen, but not exaggerated.	7. Final deflection T′ frequently much exaggerated.

Figure 84 illustrates block of the right bundle branch, and Fig. 85, of the left bundle branch.

Complex of Normal and Low Amplitude.—Aberrant Q R S complexes in all derivations of the electrocardiogram have called forth considerable discussion during the last few years. Lewis at one time ascribed these abnormal complexes to intraventricular block. In 1915 Oppenheimer and Rothschild named the condition arborization block and stated the belief that lesions involving the arborizations of the auriculoventricular bundle are responsible for the bizarre complexes. Robinson reported similar findings which resulted, he believed, from functional myocardial fatigue. Carter described sclerosis affecting the bundle branches and arborizations which he believed produced the abnormal electrocardiogram.

Recent experimental work by Smith failed to confirm the arborization hypothesis. Following coronary ligation, extensive

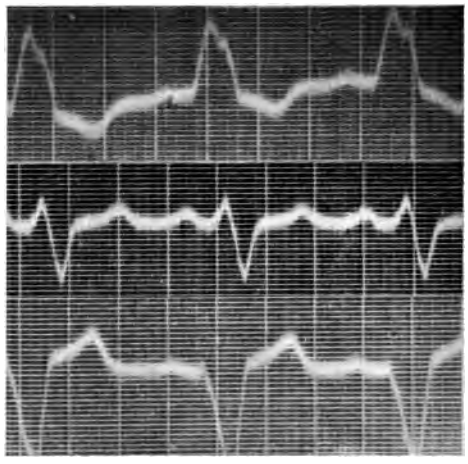

Fig. 84.—Right bundle branch block.

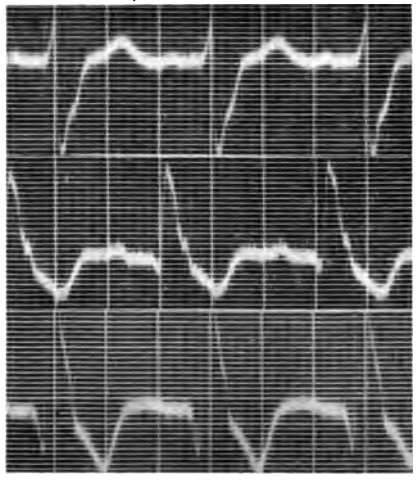

Fig. 85.—Left bundle branch block.

subendocardial lesions were produced without the development of Q R S changes. Abnormal complexes were obtained, however,

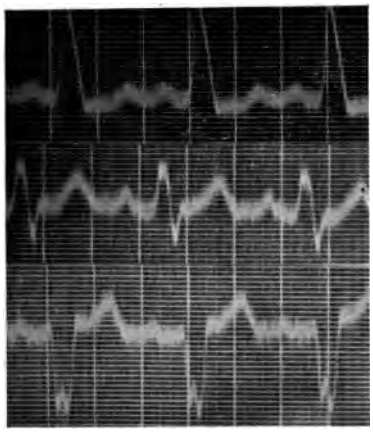

Fig. 86.—Aberrant Q R S complexes all derivations. Q R S interval 0.11 to 0.12 second. Definite notching of apex R.

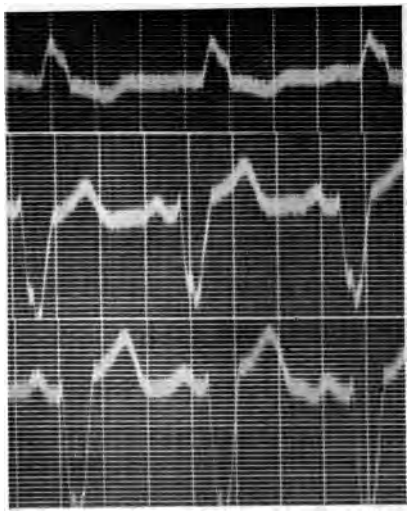

Fig. 87.—Aberrant Q R S complexes all derivations. Q R S interval 0.13 to 0.15 second. Definite notching of apex R.

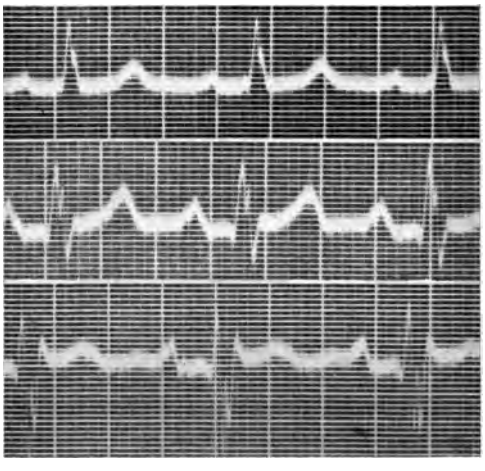

Fig. 88.—Aberrant Q R S complexes all derivations. Q R S interval 0.07 to 0.08 second. Splintering of descending limb.

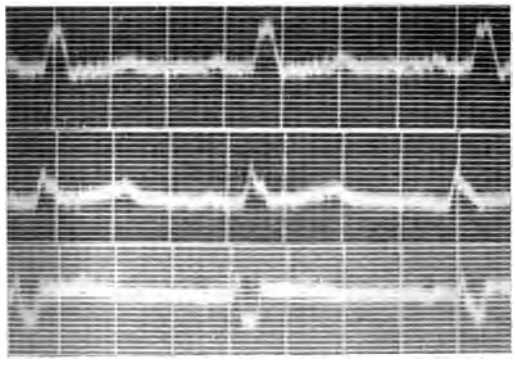

Fig. 89.—Aberrant Q R S complexes all derivations. Q R S interval 0.08 to 0.11 second. Notching of apex R.

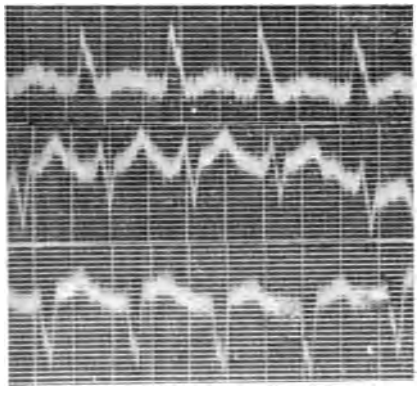

Fig. 90.—Aberrant Q R S complexes all derivations. Q R S interval 0.10 to 0.11 second. Derivation I, splintering descending limb. Derivation II, slurring of both limbs. Derivation III, notching of apex R.

Aberrant Q R S Complexes in Isolated Derivations.—Abnormalities of the Q R S complex in isolated derivations of the electrocardiogram are quite frequently observed in routine clinical electrocardiography. The aberrant complexes may be grouped into those having notching of the apex or limbs, and those in which slurring or thickening of the apex or limbs occurs. These findings obviously are less striking than the findings considered in the previous discussion.

Notched Q R S Complexes.—Wedd has called attention to the relationship, at times, of slight notching or localized thickening of the R complex to myocarditis.

I recently analyzed a series of electrocardiograms of 550 patients[40] having notching of the Q R S complex in isolated derivations; 77 (14 per cent.) were placed in Derivation I, 83 (15.1 per cent.) in Derivation II, and 390 (70.9 per cent.) in Derivation III.

Etiologic disorders in this series in order of frequency were: (1) degenerative processes 41.6 per cent., including hypertension

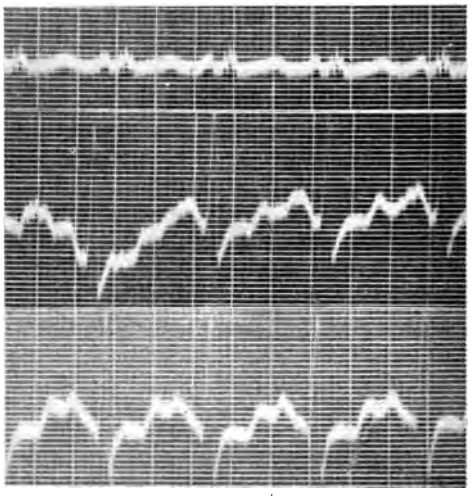

Fig. 91.—Notched Q R S complexes in Derivation I.

with and without clinical nephritis 18 per cent., exophthalmic goiter 17.6 per cent., and adenomas with hyperthyroidism 6 per cent.; (2) infections 36.9 per cent., including endocardial valvular disease 21.6 per cent., chronic myocarditis 11.3 per cent., and syphilis 4 per cent.; (3) local nutritional disturbances, including arteriosclerosis and angina pectoris 6 per cent.; and (4) congenital heart disease 0.07 per cent.

This group, collected during a period of four and one-half

years, revealed a cardiac mortality of 23.7 per cent. A control series corresponding in number, sex, and occurrence by decade and excluding the graver types of heart disease, such as angina pectoris, aneurysm, disease of the auriculoventricular

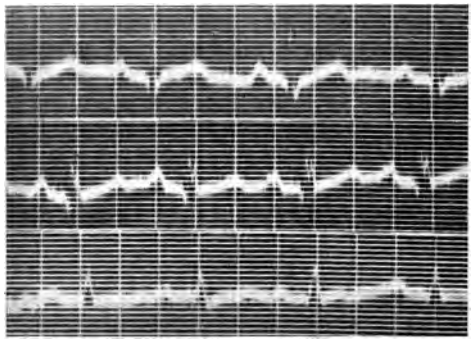

Fig. 92.—Notched Q R S complexes in Derivation II.

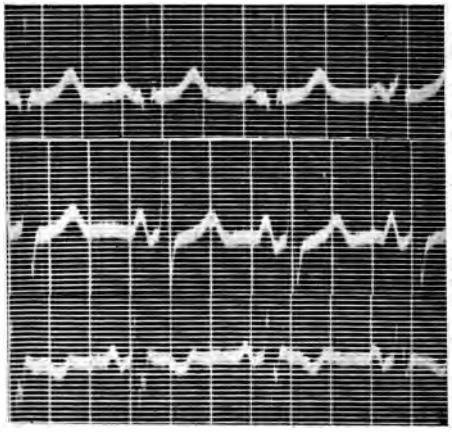

Fig. 93.—Notched Q R S complexes in Derivation III.

bundle and auricular flutter, revealed a cardiac mortality of 11.8 per cent. This difference in mortality is certainly definite (Figs. 91–93).

Slurred Q R S Complexes.—A group of electrocardiograms of 197 patients analyzed had Q R S complexes in which slurring

or localized thickening of both limbs or of the apex occurred. These changes are only slight departures from the normal: 67 cases (34 per cent.) occurred in Derivation I, 36 (18.3 per cent.) in Derivation II, and 94 (47.7 per cent.) in Derivation III.

The etiologic factors in this group were: Degenerative processes 34 per cent., including hypertension with and without clinical nephritis 17.8 per cent., exophthalmic goiter 12.7 per cent., and adenomas with hyperthyroidism 3.5 per cent.; infections 35.4 per cent., including endocardial valvular disease 22.3 per cent., chronic myocarditis 9.6 per cent., and syphilis 3.5 per cent., and

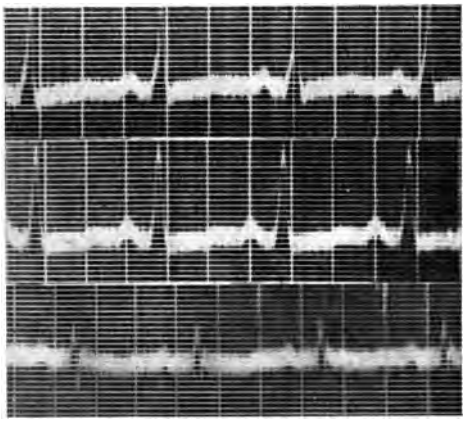

Fig. 94.—Slurred Q R S complexes in Derivation I.

local nutritional disturbances 8.1 per cent., including arteriosclerosis, angina pectoris, and congenital heart disease 1 per cent.

The cardiac mortality in this group was 24 per cent., in contrast to 14.3 per cent. in the control group.

Definite conclusions cannot be drawn with regard to notching and slurring of the Q R S complexes in isolated derivations of the electrocardiogram. The findings are at times transient, but when they occur as permanent phenomena they must be treated with due consideration (Figs. 94–96).

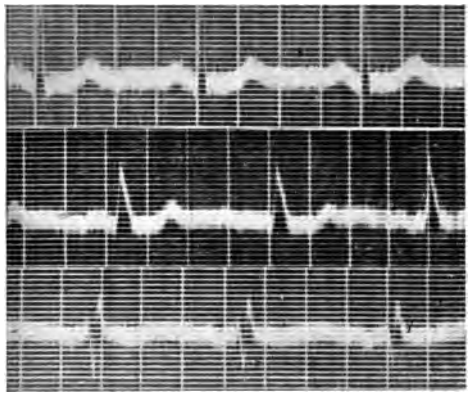

Fig. 95.—Slurred Q R S complexes in Derivation II.

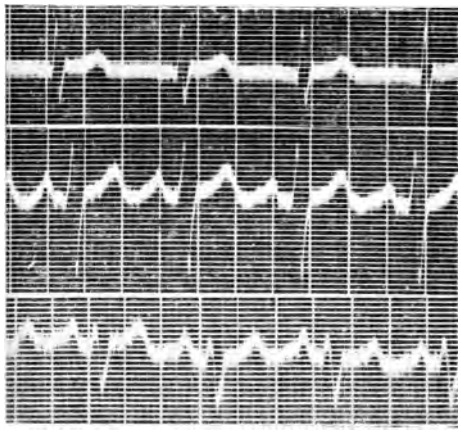

Fig. 96.—Slurred Q R S complexes in Derivation III.

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CHAPTER VIII

VENTRICULAR PREPONDERANCE AND THE ELECTROCARDIOGRAM

THE reliability of electrocardiography as a method of determining hypertrophy of the ventricles is open to serious criticism. The heart is an organ having three dimensions, and the three derivations of electrocardiography lie in a frontal plane only. Therefore, knowledge of potential of other planes is only indirectly and imperfectly obtained by projection of potential differences on the frontal plane. We have no reasons to believe that increase of muscle mass from hypertrophy is confined to one plane. This fact constitutes the greatest criticism against electrocardiography as a method for determining ventricular hypertrophy. In electrocardiography, however, we have a procedure which, within certain limits, gives us relative knowledge regarding the preponderance of one ventricle over the other.

The graphic criteria generally accepted as indicative of ventricular preponderance deal with changes in the amplitude and direction of the R wave. Preponderance of the right ventricle is supposedly indicated by a downwardly directed R wave (S) in Derivation I and an upwardly directed R wave in Derivation III. In preponderance of the left ventricle the reverse of this status obtains (Figs. 97, 98).

Einthoven, Fahr, and deWaart believe that the direction of the electric axis of the heart corresponds roughly to the anatomic axis in normal hearts at an angle varying between 40° and 90° from the horizontal. Changes were observed in the axis in mechanical displacement of the heart by posture and by respiratory movements. Calculation of their angle α determined the electric axis

of the heart, believed to be an index of ventricular balance. Angles between 40° and 90° showed a normal balance, angles above 40°

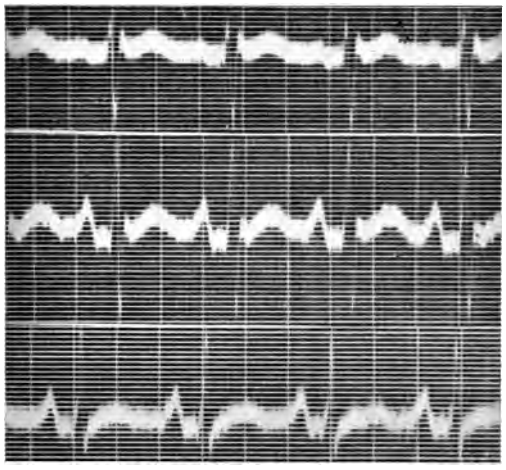

Fig. 97.—Electrocardiogram of right ventricular preponderance. R wave directed downward in Derivation I and upward in Derivation III.

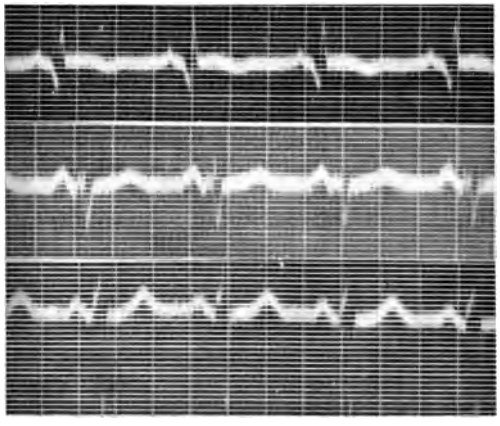

Fig. 98.—Electrocardiogram of left ventricular preponderance. R wave directed upward in Derivation I and downward in Derivation III.

showed left preponderance, and angles to the patient's right of 90°, right preponderance.

Lewis, in 1914, suggested a formula for obtaining an index of preponderance. This formula is based on the belief that the R wave in Derivation I varies with the S wave in Derivation III, and vice versa, the former diminishing and the latter increasing with preponderance of the left ventricle. Likewise the S wave in Derivation I and the R wave in Derivation III vary together in preponderance of the right ventricle.

The values for the R wave and the S wave are determined in terms of tenths of millivolts (millimeters on the electrocardiogram). $R_1 - R_3 + S_3 - S_1 =$ Index. For example, if R_1 measures 16 mm., R_3, 5 mm., S_1, 3 mm., and S_3, 22 mm., then $16 - 5 + 22 - 3 = + 30$.

Lewis was impressed by the discrepancy between clinical and electrocardiographic evidences of ventricular hypertrophy. He undertook a study of ventricular weights and called attention to errors in clinical interpretation of hypertrophy, as well as to inaccuracies of estimating hypertrophy in the heart after death. He emphasized that "preponderance" is a term used regardless of the weight of the entire heart, but it constitutes a ratio between the two ventricles which determines the form of the electrocardiogram.

Cotton likewise made a study of ventricular weights in relation to electrocardiographic determinations; his L/R weight ratios are comparable with those obtained by Lewis. L and R refer to left and right ventricular weights.

White and Bock suggested the following formula as a quantitative determination of ventricular preponderance: $(U_1 + D_3) - (D_1 + U_3) =$ Index.

U_1 is the amplitude of the greatest upward deflection of the Q R S group in Derivation I, and U_3 the greatest upward deflection in Derivation III. D_1 is the greatest downward deflection in Derivation I, and D_3 the corresponding deflection in Derivation

III. The deflection amplitudes are measured in tenths of millivolts (millimeters on the electrocardiogram). For example, if the values given in Lewis' formula are used, $(16 + 22) - (3 + 5) = + 30$. This agrees very closely with Lewis' index except in the electrocardiograms, in which the Q wave is greater than the S wave, and thus becomes the greatest downward deflection.

Carter and Greene have used the sum of the downward deflections of each derivation subtracted from the upward deflections and the resulting figures used in determining the direction of the current in the heart. This is based on the assumption that the deflection represents the resultant of opposing or balanced currents from the two sides of the heart. Einthoven's original schematic triangle was modified and employed in their calculations, since they believed that ventricular preponderance can be determined only by obtaining the direction of the electric axis.

Table 5

COMPARISON OF L R WEIGHT RATIOS AND ELECTROCARDIOGRAPHIC PREDOMINANCE DETERMINED BY FOUR METHODS

Case	Author	L R weight ratio	Angle found by Einthoven's method	Angle found by Carter's method	Lewis' formula	White's index
1	Cotton	2.63	- 10	- 30	29.5	29.5
2	Lewis	2.60	- 70	- 60	19.0	19.0
3	Cotton	2.55	- 74	- 75	17.5	16.5
4	Lewis	2.04	14	16	19.0	19.0
5	Cotton	1.97	79	75	-10.0	-8.0
6	Lewis	1.94	45	30	8.0	8.0
7	Lewis	1.87	0	10	12.5	12.5
8	Lewis	1.82	18	35	4.5	4.5
9	Lewis	1.60	82	115	-7.0	-7.0
10	Cotton	1.55	4	-30	9.0	9.0
11	Lewis	1.38	78	90	1.5	1.5
12	Cotton	1.30	79	95	-3.5	-5.0
13	Cotton	0.82	101	150	-6.0	-6.0
14	Lewis	0.82	105	128	-15.5	-15.5
15	Lewis	0.41	131	160	-77.0	-76.0

Pardee, after a careful comparison of the various methods advocated for determining ventricular preponderance by means of the electrocardiogram, found gross discrepancies. Table 5

is modified from Pardee. He points out that formulas hold true only in a very general way, since individual electrocardiograms vary from the position in the scale of ventricular preponderance as determined by ventricular weights. He further discusses the element of error arising from variations in the position and type of the heart.

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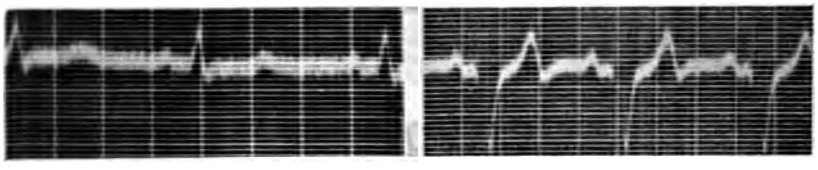
CHAPTER IX

ABNORMALITIES OF THE T WAVE

As I have stated in Chapter III, the T wave is a contraction phenomenon resulting from changes in contraction preponderance on one side of the line of equipotential. Abnormalities of the T wave result from changes in electropotential produced by changes in contraction preponderance.

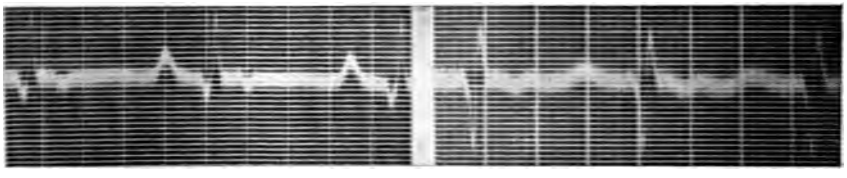
POSITIVE T WAVE OF HIGH AMPLITUDE

The abrupt peaked T wave of high amplitude is not infrequently encountered in routine clinical electrocardiography; it is



A B

Fig. 99.—A, Usual type of T wave; rounded, low amplitude. B, Abnormal T wave; abruptly peaked, high amplitude.



A B

Fig. 100.—A, Rounded, low amplitude negative T wave. B, Abruptly peaked, high amplitude negative T wave.

in sharp contrast to the usual blunt curved wave of lower amplitude (Figs. 99, 100). It is present at times in a single derivation, but

more often in combined derivations. Such a T wave is often observed in hyperthyroidism before myocardial failure has occurred. It is present in about 10 per cent. of patients having angina pectoris. The wave is evidence, I believe, of rather sudden changes in contraction preponderance, indicative probably of increased contraction of certain muscular areas. An analysis of Smith's electrocardiograms taken after ligation of the coronaries in dogs reveals numerous instances of abrupt peaked positive T waves of high amplitude.

T WAVE NEGATIVITY

Many opinions have been expressed with regard to the significance of negativity or inversion of the T wave in isolated or combined derivations of the electrocardiogram.

Cohn, Fraser, and Jamieson have called attention to negativity of the T wave after the administration of digitalis. This effect of digitalis has been ascribed to muscular ventricular redistribution, or possibly to alteration in muscular contractility; the changes are not permanent.

Numerous statements may be found in which myocardial damage is ascribed to T wave negativity in certain derivations, and again, these occurrences have been noted in apparently normal hearts affecting, largely, Derivation III.

Smith, during his experimental work on coronary ligations, observed interesting changes in the T wave. The most constant changes in the electrocardiogram following ligation of any branch of the left coronary artery affected the T wave. A strongly positive to a markedly negative wave resulted fairly constantly with a slower return to positive or iso-electric. The negativity was usually observed within twenty-four hours after ligation and lasted for from three to four days. The duration seemed to bear a relationship to the size of the artery ligated. This work offers a tangible basis in directing attention to changes in the intrinsic blood-supply

of the heart, as evidenced by greater or lesser derangements of cardiac function. Morison has shown that blood-volume alterations may produce changes detectable in the electrocardiogram.

Consideration of Electropotential.—Waller and Reid demonstrated a line of equipotential passing through the heart from the base to the apex in relation to any two derivations from the extremities. A preponderance of negativity above this line, representing the cardiac base, caused deflection of the galvanometer connected to both upper extremities in a manner to indicate relative negativity of the electrode connected with the right arm. The arm becomes relatively negative in derivations from an arm and a leg. Dominance of negativity below the equipotential line deflects the galvanometer in the opposite direction.

Hypothesis of T Wave Negativity.—The three derivations of the electrocardiogram possess symbols of definite electropotential in relation to their electrodes. In the normal electrocardiogram symbols are as follows: Derivation I, right arm −, left arm +; Derivation II, right arm −, left leg +; and Derivation III, left arm −, left leg + (Fig. 101). This arrangement implies positive or upright deflections in all derivations of the electrocardiogram. If it is assumed that the "conduction-contraction" theory is correct the T wave is the expression of preponderance of contraction on one side of the line of equipotential. T wave negativity (inversion), therefore, results from changes in contraction preponderance. The negativity of this wave in certain isolated or combined derivations of the electrocardiogram is indicative of definite potential changes affecting contraction preponderance in various regions of the cardiac musculature.

In the normal heart, therefore, according to standard derivations the T wave in all derivations is positive (upright), the upper right zone of potential is strongly electronegative to the apical zone, while the left upper zone is iso-electric. This potential

Positive T wave in all Derivations (normal)

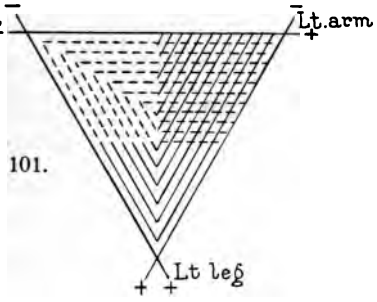


Fig. 101.

Negative T wave in Derivation I.

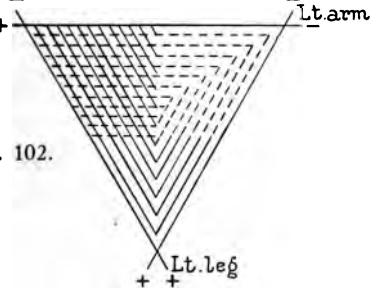


Fig. 102.

Negative T wave in Derivation III.

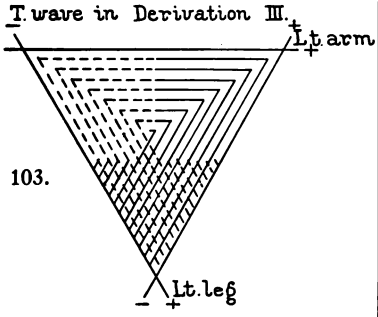


Fig. 103.

Negative T wave in Derivations I and II.

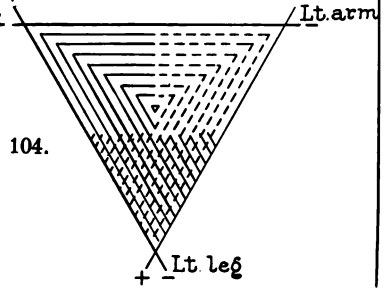


Fig. 104.

Negative T wave in Derivations II and III

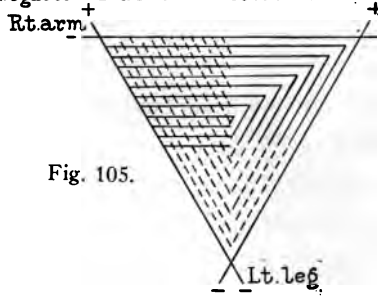


Fig. 105.

Negative T wave in Derivations I, II and III

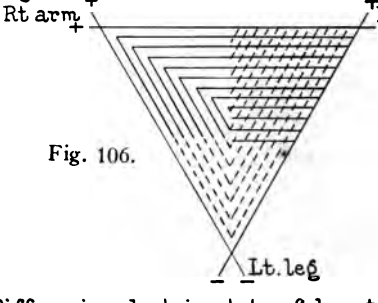


Fig. 106.

Diffuse iso-electric state of heart if negative T. wave in Derivation II occurred.

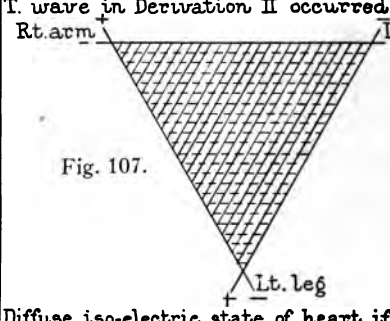


Fig. 107.

Diffuse iso-electric state of heart if negative T. wave in Derivations I and III occurred.

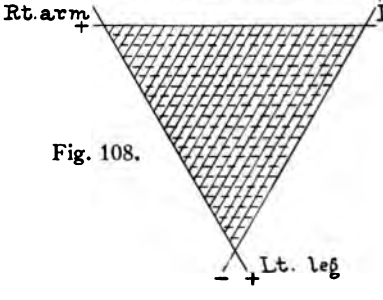


Fig. 108.

Figs. 101–108.—Observations on negativity of the final ventricular *T* wave of the electrocardiogram.

arrangement is illustrated in Fig. 101. For reasons of simplification I have represented the three derivations by the sides of an equilateral triangle. To prevent misunderstanding it should be stated that the schematic figure employed, divided into zones of electropotential, is not based on mathematic consideration. The right upper zone in general corresponds to the sinus region of the heart which is the seat of primary negativity.

Changes in the normal potential distribution produce T wave negativity in isolated or combined derivations of the electrocardiogram. Reversal of potential in one derivation alters cardiac potential so that T wave negativity in that derivation occurs.

Increased general cellular function implies increased blood volume for the maintenance of normal tissue metabolism. In a specialized organ this augmentation is manifested by an increase in its function. In the heart, increase in the blood volume beyond physiologic limits increases contraction. I refer particularly to increase of ventricular volume, since coronary volume is largely dependent on this factor. Because of impairment or disease of certain intrinsic channels of blood-supply the affected muscle does not receive the requisite amount of blood properly to maintain function, while the unaffected muscle demands greater blood volume for relatively more efficient contraction. This is an explanation for T wave changes in isolated or combined derivations occurring permanently or temporarily.

The Action of the Cardiac Nerves on T Wave Negativity.—Samojloff and Dale and Mines were able to produce T wave negativity by stimulation of the cardiac vagus.

Rothberger and Winterberg made the same observation following stimulation of the left cervical sympathetic branches. A series of clinical observations was conducted on patients having negative T waves in isolated or combined derivations of their electrocardiograms. Following the initial tracing, pressure was ap

to the right vagus region in the neck, to the left vagus region, and finally to the right eyeball (oculocardiac reflex) and the respective electrocardiograms obtained. In no instance was a change noted in the negative T wave or in the positive T wave of the unaffected derivations. Atropin (gr. $\frac{1}{150}$) was then administered subcutaneously and records obtained every ten minutes for forty minutes. Again no effect on the T wave was noted. No change occurred following the subcutaneous administration of epinephrin (0.5 c.c. of 1 : 1000 solution). These clinical procedures, however, are obviously not so accurate as direct experimental stimulation.

There has been no adequate explanation of the occurrence of the negative T wave in isolated or combined derivations of the electrocardiogram. In the Mayo Clinic we have repeatedly observed the negative T wave in Derivations I and III, in combined Derivations I and II, in combined Derivations II and III, and in combined Derivations I, II, and III. No instances of negativity in Derivation II or in combined Derivations I and III are recorded. This observation in 7000 electrocardiographic examinations eliminates the element of coincidence.[10] There is a definite reason why these changes do not occur. Lewis states that the T wave is always upright in Derivation II alone.

T Wave Negativity in Derivation I.—Figure 102 represents the arrangement of potential in this abnormality. The left arm becomes electronegative to the right, while the other signs remain unchanged. The right upper zone, instead of being electronegative, becomes iso-electric, while the left upper becomes electronegative to the apex. The occurrence of an iso-electric state in the right upper zone is a marked departure from normal in that the area of primary electronegativity is altered.

T Wave Negativity in Derivation III.—The left leg becomes electronegative to the left arm; the other derivations remain unchanged. The right upper zone remains electronegative with

reference to the left upper. The apical zone becomes iso-electric. This distribution is but a slight deviation from normal in that the right upper zone remains electronegative (Fig. 103).

T Wave Negativity in Combined Derivations I and II.—The left arm becomes electronegative to the right arm, and the left leg electronegative to the right arm. The third derivation remains unchanged. The left upper zone becomes electronegative to the right zone, and the apical zone becomes iso-electric. This arrangement again deviates from the normal in altering the area of primary electronegativity; it is a lesser change in that the right upper zone becomes electropositive instead of iso-electric.

T Wave Negativity in Combined Derivations II and III.—The left leg becomes electronegative to both arms. Derivation I remains unchanged. The apical zone becomes electronegative to the left upper zone, while the right upper zone becomes iso-electric. This arrangement, as in Derivation I, is a marked change from normal in that the area of primary electronegativity becomes iso-electric (Fig. 105).

T Wave Negativity in Combined Derivations I, II, and III.—All derivations reverse their signs in relation to each other. The apical zone becomes electronegative to the right upper zone, while the left upper zone becomes iso-electric. The upper zones assume just the opposite relationship to each other that occurs in combined Derivations II and III. Here again the potential distribution is disturbed, the area of primary electronegativity becoming electropositive, approaching the changes accompanying T wave negativity in combined Derivations I and II (Fig. 106).

Figures 107 and 108 illustrate the reason why T wave negativity in Derivation II and in combined Derivations I and III does not occur. In Derivation II the left leg would be electronegative to the right arm, while the other derivations would remain unchanged. This distribution would imply a diffuse iso-electric cardiac state

and would indicate that cardiac activity had ceased. In combined Derivations I and III the left arm would be electronegative to the right arm, and the left leg electronegative to the left arm. This arrangement likewise would indicate a diffuse iso-electric state.

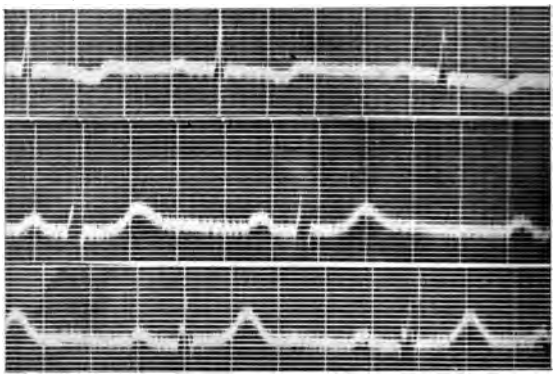


Fig. 109.—Negative T wave in Derivation I.

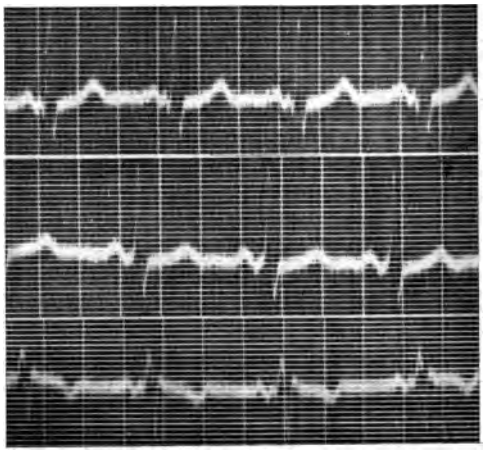


Fig. 110.—Negative T wave in Derivation III.

The greater the deviation from the normal potential distribution, the greater the significance of the disorder responsible for the change. The greatest change which is compatible with life is the

iso-electric state occupying the right upper zone. Therefore T wave negativity in Derivation I and in combined Derivations II and III should be associated generally with grave heart disease.

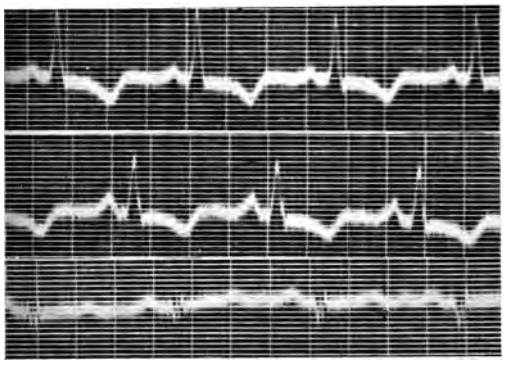


Fig. 111.—Negative T wave in Derivations I and II.

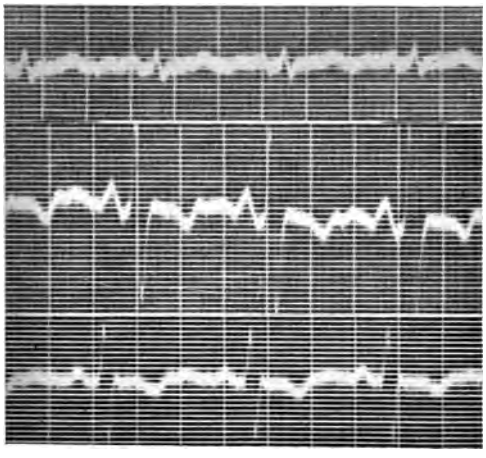


Fig. 112.—Negative T wave in Derivations II and III.

Next in significance should be the distributions of potential, rendering the left upper zone electronegative to the right, and represented by T wave negativity in combined Derivations I and II and in combined Derivations I, II, and III. In the distribution which

most closely approximates normal the T wave is negative in Derivation III. In these cases the normal potential relationship of the right upper zone is maintained (Figs. 109–113).

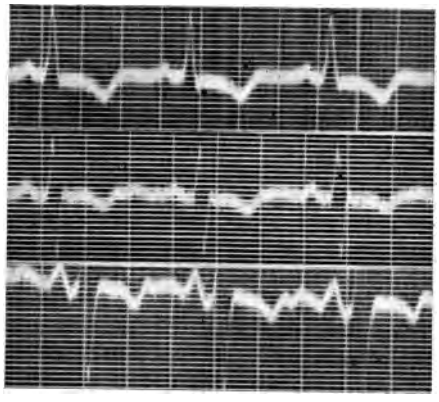


Fig. 113.—Negative T wave in Derivations I, II, and III.

Clinical Consideration of T Wave Negativity.—I have recently completed an analysis of 1106 cases of T wave negativity in the electrocardiograms[10]: 140 instances (12.6 per cent.) were noted in Derivation I, 688 (62.2 per cent.) in Derivation III, 62 (5.6 per cent.) in combined Derivations I and II, 171 (15.5 per cent.) in combined Derivations II and III, and 45 (4.1 per cent.) in combined Derivations I, II, and III. There was no instance of T wave negativity in Derivation II nor in combined Derivations I and III. Patients who had had digitalis within six weeks of the time of electrocardiographic examination were not included in this series.

DISEASES ASSOCIATED WITH T WAVE NEGATIVITY

Derivation I (140 Cases).—Myocardial degeneration associated with the hypertension group occurred with greatest frequency (38.6 per cent.) in the patients having T wave negativity in Derivation I of their electrocardiograms. Chronic endocardial valvular

disease occurred second in order of frequency (22.1 per cent.). Local nutritional disturbances including arteriosclerosis and angina pectoris occurred in 13.6 per cent. of the cases. In no instance was the cardiac examination negative. In the majority of instances grave heart disease was present; 53 patients had aberrant Q R S complexes in all derivations of their electrocardiograms, 3 had delayed auriculoventricular conduction, 3 had complete auriculo-ventricular dissociation, 19 had auricular fibrillation, 1 had auricular flutter, and 1 had ventricular tachycardia; 16 patients had aortic disease; 9 had angina pectoris.

The high incidence of grave heart disease in this group verifies my previous statement regarding the potential distribution responsible for this negativity as being the greatest departure from normal. The right upper zone is iso-electric instead of electronegative.

Derivation III (688 Cases).—The relative frequency of T wave negativity occurring in Derivation III is apparent. The myocardial degeneration secondary to exophthalmic goiter was the most frequently associated condition (19.2 per cent.). Chronic endocardial valvular disease occurred in 16.9 per cent., and chronic myocarditis in 14.8 per cent. Myocardial degeneration associated with the hypertension group occurred in only 10.8 per cent. of the cases.

Grave heart disease is relatively infrequent in this group; 3 patients had aberrant Q R S complexes in all derivations of their electrocardiograms, 2 had delayed auriculoventricular conduction, and 11 had auricular fibrillation; 8 patients had aortic disease, and 21 had angina pectoris; 20 per cent. of the patients had no demonstrable evidence of organic heart disease. Of this number, 40 per cent. had cardiac neurosis. The relative infrequency of grave heart disease and the high percentage of apparently normal hearts are in marked contrast to the findings associated with T wave negativity in Derivation I. These findings are in accord

with the hypothetic significance of T wave negativity in Derivation III.

Combined Derivations I and II (62 Cases).—Myocardial degeneration associated with hypertension occurred in 50 per cent. of the patients having T wave negativity in combined Derivations I and II. Chronic endocardial valvular disease was present in 20.9 per cent., and chronic myocarditis in 11.3 per cent. of the cases. Local nutritional disturbances, including arteriosclerosis and angina pectoris, occurred in 8.1 per cent. of the cases. About half (46.7 per cent.) of the cases were associated with grave heart disease; 14 patients had aberrant Q R S complexes in all derivations of their electrocardiograms, 2 had delayed auriculoventricular conduction, 5 had auricular fibrillation, and 1 patient had ventricular tachycardia; 6 patients had aortic disease, and 1 of these had an aortic aneurysm. One patient had angina pectoris.

Combined Derivations II and III (171 Cases).—Chronic endocardial valvular disease occurred with greatest frequency (26.9 per cent.) in those patients having T wave negativity in combined Derivations II and III. In order of frequency followed myocardial degeneration associated with exophthalmic goiter (21 per cent.), chronic myocarditis (17.5 per cent.), and myocardial degeneration associated with hypertension (15.8 per cent.).

A large percentage of the patients had grave heart disease; 11 patients had aberrant Q R S complexes in all derivations, 3 had delayed auriculoventricular conduction, 1 patient had complete auriculoventricular dissociation, 24 patients had auricular fibrillation, and 1 patient had ventricular tachycardia; 13 patients had aortic disease, and 3 had angina pectoris. In 9 cases the cardiac examination was negative.

Combined Derivations I, II, and III (45 Cases).—T wave negativity in combined Derivations I, II, and III constituted the smallest group, 4.1 per cent of the total series. Myocardial degen-

eration associated with the hypertension group occurred most often (35.5 per cent.), and in order of occurrence, chronic endocardial valvular disease (24.4 per cent.) and chronic myocarditis (20 per cent.). Every patient in this group had definite clinical evidence of heart disease.

Three of the patients with the graver forms of heart disease had aberrant Q R S complexes in all derivations, 3 had delayed auriculoventricular conduction, and 11 had auricular fibrillation; 8 patients had aortic disease, and 2 had angina pectoris.

CARDIAC MORTALITY AND T WAVE NEGATIVITY

Derivation I.—Information has been received concerning 117 patients having T wave negativity in Derivation I of their electrocardiograms: 78 (66.6 per cent.) have died from heart disease during a period of four and one-half years; 33 patients are alive; 10 of these report their conditions worse, 15 improved, and 8 unchanged. None of the patients was without cardiac complaints.

The high cardiac mortality in this group is in accord with the hypothetic and clinical significance, which I have emphasized, as attending this T wave negativity. To prevent misunderstanding it may be stated that the negative T wave, *per se*, is only the manifestation of serious underlying myocardial disorder. Changes in cardiac function affecting contraction preponderance resulting from organic or functional myocardial fatigue alter electropotential, which produces T wave negativity.

Derivation III.—Information has been received concerning 487 patients having T wave negativity in Derivation III of their electrocardiograms; 46 (9.4 per cent.) have died from heart disease during a period of four and one-half years. This relatively low cardiac mortality is sharply contrasted with the mortality in the foregoing group. It is in agreement, however, with the hypothetic and clinical significance accorded this T wave n

potential distribution producing this negativity is but a slight departure from normal: 420 patients are alive; 97 report their conditions worse, 161 improved, and 162 unchanged. One hundred and six patients report no cardiac complaints.

Derivations I and II.—Of the 52 patients having T wave negativity in combined Derivations I and II concerning whom information has been received, 35 (67.3 per cent.) have died from heart disease during four and one-half years. This mortality is greater than was anticipated, since the potential distribution resulting in this T wave negativity was not the greatest departure from normal. The fact that the group is relatively small may be a factor in obtaining a high cardiac mortality; 16 patients are alive; 1 patient reports his condition worse, 12 patients are improved, and 3 unchanged. Only 1 patient reports no cardiac complaint.

Combined Derivations II and III.—We have learned of the condition of 135 patients having T wave negativity in combined Derivations II and III: 35 (25.9 per cent.) have died from heart disease during a period of four and one-half years. In contradistinction to the foregoing group, the mortality was lower than expected from a hypothetic consideration of potential distribution and associated heart disease. A possible explanation for this discrepancy rests in the fact that 23.3 per cent. of the patients were examined during the last year of the series, and the time element, therefore, is too short to embrace a true mortality average; 88 patients are alive, 25 report their conditions worse, 37 improved, and 26 unchanged; 16 patients report no cardiac complaints; 7 of these had had thyroidectomies for hyperthyroidism and were cured.

Combined Derivations I, II, and III.—Patients having T wave negativity in all derivations comprised a relatively small group; 19 of the 38 (50 per cent.) on whom we have had reports have died from heart disease during four and one-half years. This

mortality agrees fairly well with hypothetic considerations of potential distribution and associated grave heart disease; 15 patients are alive. 5 report their conditions worse. 8 improved, and 2 unchanged. No patient was without cardiac complaint.

In the complete series of T wave negativity, regardless of derivation grouping, the cardiac mortality was 25.6 per cent. The mortality according to derivation grouping was: combined Derivations I and II 67.3 per cent.; Derivation I 66.6 per cent.; combined Derivations I, II, and III 50 per cent.; combined Derivations II and III 25.9 per cent., and Derivation III 9.4 per cent.

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CHAPTER X

ABNORMALITIES OF THE P WAVE

THE auricular P wave, as I have stated in Chapter III, results from contraction of the auricle and from passage of the impulse through the auricle. The normal wave is rather peaked, of low amplitude, and upright (positive).

EXAGGERATED AMPLITUDE OF THE P WAVE

The P wave is at times found to be of increased amplitude, even exceeding the amplitude of the T wave. This finding is usually present in Derivations II and III. White believes that

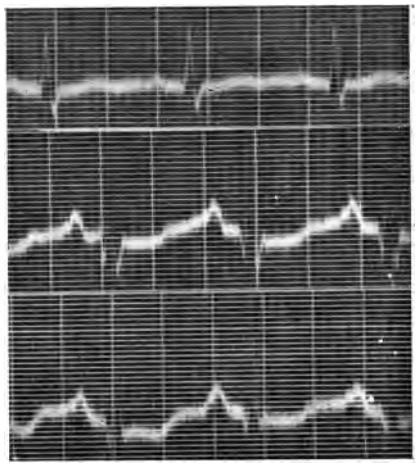

Fig. 114.—Exaggerated P wave in Derivations II and III.

P waves more than 3 by 10^{-4} millivolts in amplitude or more than 0.1 second in duration almost always indicate auricular hypertrophy. This statement requires modification, as I have observed the exaggerated P wave in hyperthyroidism when necropsy has

failed to reveal hypertrophy of the auricles. Increase in auricular activity through increase in rate or increase in contraction amplitude is likewise capable of producing the exaggerated wave. It is frequently observed in mitral stenosis, in hyperthyroidism, and in neurotic individuals with tachycardia (Fig. 114).

NOTCHING OF THE P WAVE

At times distinct notching of the apex or the descending portion of the P wave is observed, usually in those waves of exaggerated amplitude and involving particularly Derivation II or III. Such

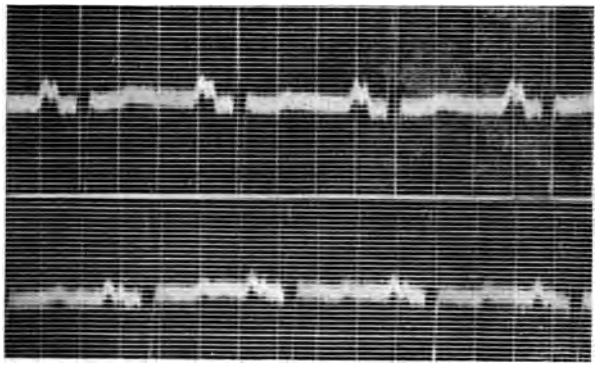

Fig. 115.—Notched P wave in Derivations II and III.

a wave probably results from auricular asynchronism. von Hoesslin observed notching of the P wave during vagus pressure in man. Weil attributed the notching to delayed conduction in the sinus node, or between the node and auricle, and believed that the first portion of the wave was the manifestation of sinus activity. The notched P wave is frequently observed in mitral stenosis (Fig. 115).

NEGATIVE P WAVE (INVERTED)

Negativity of the P wave in all derivations of the electrocardiogram is invariably evidence of an ectopic rhythm. Ritchie, Einthoven, Fahr and deWaart, von Hoesslin, and Wilson ascribed

negativity of the P wave to a change in the cardiac pace-maker, as in auricular tachycardia, flutter, and so forth.

Carter and Wedd, directing their attention to negative and diphasic P waves in Derivation III of the electrocardiogram, concluded that the negative P waves in Derivation III fall into two groups: (1) waves in which there may be a variation in the site of the pace-maker in its relationship to Derivation III, especially under the influence of the vagi and digitalis, and (2) waves in which the relationship of the pace-maker to Derivation III is

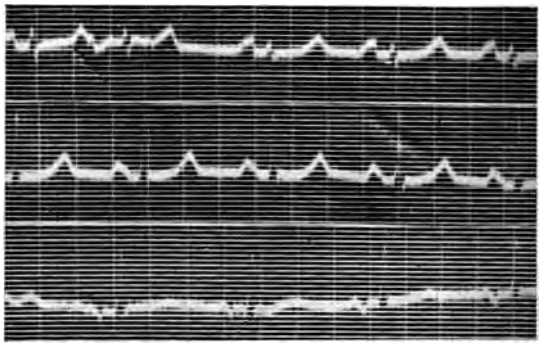

Fig. 116.—Inverted (negative) P wave in Derivation III.

such that a constantly negative P wave is absolutely not influenced by atropin, vagus pressure, or digitalis. This is possibly owing to an alteration of the muscle balance which changes the relation of the pace-maker to the axis for this derivation, or possibly to the anatomic distribution of the vagal fibers.

During routine clinical electrocardiography the negative P waves confined to Derivation III are occasionally encountered and frequently are transient phenomena (Fig. 116).

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CHAPTER XI

CONGENITAL HEART DISEASE

CONGENITAL anomalies of the heart are of interest to the clinician partly because of their infrequent occurrence, but largely because of the problems of diagnosis.

Classification of congenital cardiac defects is difficult; the most comprehensive grouping, based on anatomic considerations, is that of Hirschfelder:

I. Malformations about the heart:
   1. Malformations of the chest wall (ectopia cordis)
   2. Malformations of the pericardium

II. Abnormalities in the position of the heart:
   1. Heart on the right side (dextrocardia or dexiocardia)
   2. Position of all the organs inverted (situs transversus)
   3. Heart situated in the neck (cervical heart)
   4. Heart situated within the peritoneal cavity (abdominal heart)

III. Abnormalities of the valvular orifices:
   1. Pulmonary stenosis or atresia
   2. Supernumerary or defective cusps of pulmonary valves
   3. Tricuspid stenosis or insufficiency; malformation of the valve
   4. Aortic stenosis; atresia of the aorta; malformations of the aortic valve
   5. Mitral stenosis; malformation of the mitral valve

IV. Defects in the septa:
   1. Interventricular septum:
      (*a*) In the septum membranaceum
      (*b*) In the muscular part of the septum (below)
   2. Interauricular septum:
      (*a*) Defect or absence of valve of the foramen ovale
      (*b*) Valve normal, but not closed
      (*c*) Defect between the muscle strands in the lower portion of interauricular septum

V. Abnormalities in the cavities:
   1. Supernumerary septa
   2. Cor biatriatum trioculare
   3. Cor biloculare
   4. Cor biventriculatum triloculare
   5. Bifid apex
   6. Double heart

VI. Deviations of the septum cordis with transposition of vessels
VII. Persistence of ductus Botalli
VIII. Abnormalities of the aorta:
   1. Coarctation of the aorta:
      (*a*) Above the ductus arteriosus
      (*b*) Below the ductus arteriosus
   2. Hypoplasia of the aorta
   3. Malformations of the aortic arch
IX. Abnormalities in the arrangement and formation of the veins

The clinical identification of the types of lesions in congenital heart disease is difficult and possible only within certain limits. Diagnostic accuracy is further made more difficult by the frequent association of multiple anomalies. All of the electrocardiograms in this chapter are, therefore, not based on proved lesions, but on diagnostic probability; absolute identification of congenital defects must be reserved for the pathologist.

CONGENITAL DEXTROCARDIA (RIGHT-SIDED HEART)

Two types of congenital dextrocardia are recognized: one associated with transposition of the abdominal viscera (situs transversus), and the other, associated with transposition of the heart and great vessels only. At times anomalous arrangement of the vena cava permits the admixture of arterial and venous blood, giving rise to a clinical picture simulating the syndrome of congenital heart disease.

By recalling the embryologic development of the heart it is readily understood how transposition of this organ occurs. The two primitive cardiac tubes fuse into one about the fifteenth day, and auricular, ventricular, and bulbar subdivisions become

evident. The tube soon becomes bent on itself, which determines largely the future axis of the heart. In congenital transposition the primitive tube bends into a contrasigmoid (Ƨ) instead of the normal sigmoid (S) manner. Abbott has explained this by assum-

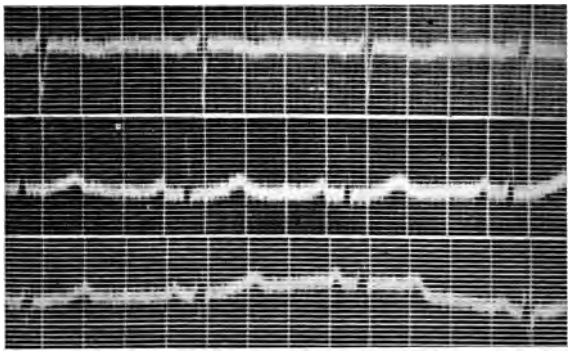

Fig. 117.—Electrocardiogram of congenital dextrocardia. Inversion of all waves in Derivation I.

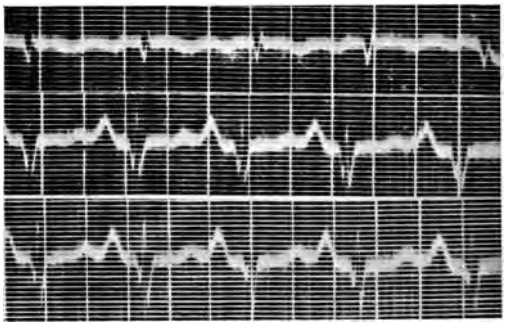

Fig. 118.—Electrocardiogram of congenital dextrocardia. Inversion of all waves in Derivation I.

ing that the embryo lies in an abnormal position within the chorion, so that its right side instead of its left lies closer to the blood-supply. I have observed 5 patients with congenital dextrocardia, in every instance associated with situs transversus. None of the patients had any complaint referable to the abnormality.

The electrocardiograms in congenital dextrocardia are characteristic, showing inversion of all the waves in Derivation I (Figs. 117, 118). Electrocardiograms of congenital dextrocardia associated with situs transversus have been published by Hoke, Nicolai, Owens, Lewis, and Willius. I have observed one patient with inversion of all waves in Derivations I and II, resulting probably from exaggeration of the inclination of the cardiac axis to the right (Fig. 119).

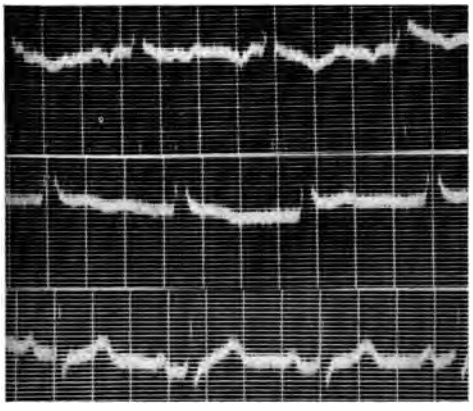

Fig. 119.—Electrocardiogram of congenital dextrocardia. Inversion of all waves in Derivations I and II.

Neuhof has observed that the R wave in Derivation III had greater amplitude than in Derivation II, but in the patients observed in the Mayo Clinic no constancy in this finding occurred.

CONGENITAL PULMONIC STENOSIS

Two causes have been ascribed to congenital valvular defects: defective development and endocarditis in fetal life. Pathologic study of cases permits credence to both views. The narrowing of the pulmonary artery may occur (1) in the trunk of the vessel between the point of ramification and the valvular ring, (2) at the valve itself from defective development of those structures

as fusion of the leaflets, and (3) below the valves and within the infundibulum of the right ventricle.

Pulmonic stenosis obviously results in stasis within the right ventricle and, therefore, this chamber bears the brunt of the burden. In 80 per cent. of the cases pulmonic stenosis is associated with patency of the interventricular septum which markedly aids circu-

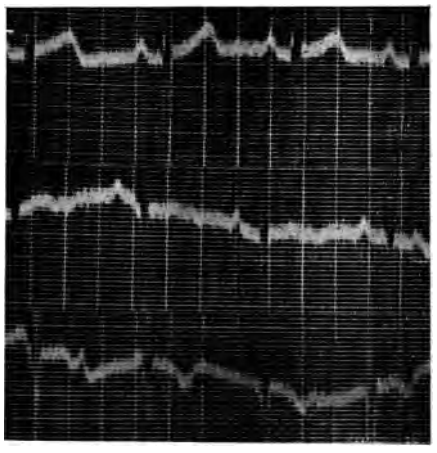

Fig. 120.—Electrocardiogram of patient with congenital pulmonic stenosis Marked preponderance of the right ventricle.

lation. The increased work of the right ventricle leads to hypertrophy, and the electrocardiograms of congenital pulmonic stenosis reveal marked preponderance of this chamber (Fig. 120). No characteristic electrocardiographic findings occur.

CONGENITAL AORTIC STENOSIS

Congenital aortic stenosis is a rare anomaly. The stenosis may occur at the valvular orifice or in the arch of the aorta, above or below the ductus arteriosus (Botalli). This lesion may or may not be associated with patency of the ductus arteriosus. The association with pulmonic stenosis and with patent interventricular septum sometimes occurs. In this anomaly, as in all aortic lesions,

the left ventricle bears the brunt of the burden, and hypertrophies. The electrocardiograms are not characteristic, preponderance of the left ventricle being present (Fig. 121).

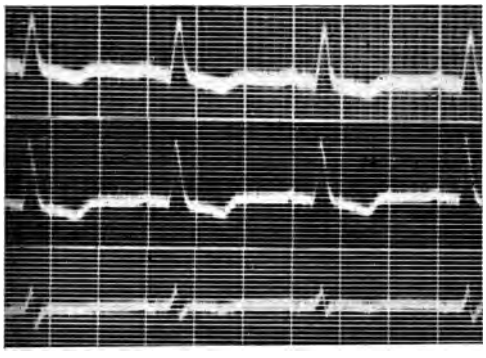

Fig. 121.—Electrocardiogram of patient with congenital aortic stenosis. Negative T wave in Derivations I, II, and III. Left ventricular preponderance.

PATENT FORAMEN OVALE

Patency of the foramen ovale is often not attended by signs and symptoms; its presence is determined by the size of the open-

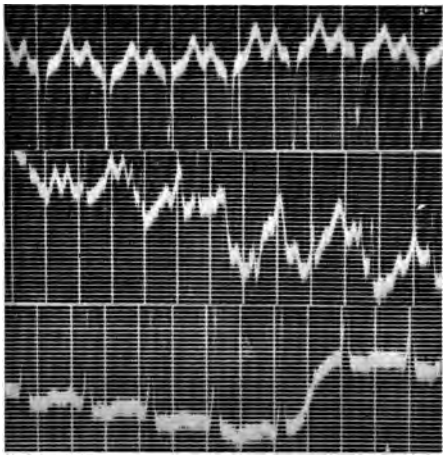

Fig. 122.—Electrocardiogram of patient with patent foramen ovale. Marked preponderance of the right ventricle. Respiratory variation of amplitude of R waves especially noticeable in Derivation I.

ing and association with other congenital cardiac defects. When the opening in the interauricular septum is small, little or no effect on the circulation is exerted. When the opening is large, and especially when the left ventricle fails or the pressure in the left auricle rises, as in mitral stenosis, the blood-current regurgitates into the right auricle. This obviously increases the work of the right heart, and frequently the electrocardiograms of these patients reveal preponderance of the right ventricle (Fig. 122).

INTERVENTRICULAR SEPTUM DEFECT

Defects of the interventricular septum uncomplicated by other congenital anomalies are rare. Pulmonic stenosis is the usual

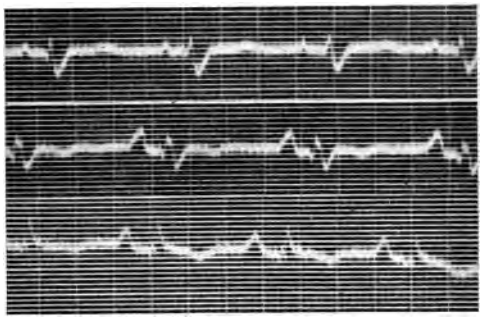

Fig. 123.—Electrocardiogram of patient with congenital interventricular septum defect. Notched Q R S complexes in Derivations I and II. Exaggerated P wave in Derivations II and III. Right ventricular preponderance.

associated defect. If the defect is associated with pulmonic stenosis, the blood is allowed to pass from the right to the left ventricle, relieving the stress of the former and increasing the work of the latter. In the uncomplicated defect of the septum the stress of both ventricles may be equalized and remain so until one or the other begins to fail from some adventitious cause. It is, therefore, evident that the resulting electrocardiograms may show either right or left ventricular preponderance, and in some instances no imbalance (Fig. 123).

Fig. 178. Electrocardiogram of patient with congenital patent ductus arteriosus (Botalli) and pulmonary stenosis. Note the auriculoventricular dissociation with ventricular rate exceeding that of the auricles. P wave negative. Segregation in the auricle of the T wave.

PATENT DUCTUS ARTERIOSUS (BOTALLI)

Patency of the ductus arteriosus is frequently associated with pulmonic stenosis. Any condition during fetal life which interfered with the flow of blood through the aorta favors the persistence of the ductus arteriosus. Hypertrophy of the right ventricle is the rule.

Figure 124 is the electrocardiogram of a boy having a patent ductus arteriosus associated with pulmonic stenosis. Complete

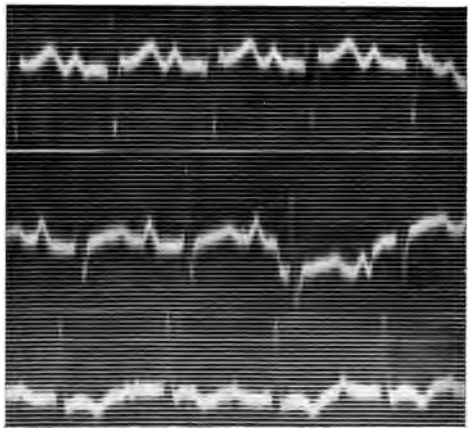

Fig. 125.—Electrocardiogram of patient with patent ductus arteriosus (Botalli). Preponderance of the right ventricle.

auriculoventricular dissociation is present and an unusual mechanism resulting in a ventricular rate greater than the rate of the auricles. This is due to a constant progression of the auricular impulse which is ectopic (Fig. 125).

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CHAPTER XII

ELECTROCARDIOGRAPHIC STUDY OF MISCELLANEOUS HEART DISEASES

ELECTROCARDIOGRAPHY, as I have stated, is dependent on electropotential resulting from cardiac activity, and this potential is developed in the cardiac musculture. Electrocardiograms of various types of heart disease, therefore, deviate from the normal only as the muscle-mass is affected, whereby electropotential becomes altered. Since changes in potential may result from organic or functional myocardial changes, it is obvious that pericarditis, aneurysm, and innumerable other diseases do not result in characteristic electrocardiograms. Certain conditions, however, reveal tracings with certain uniformities. Some of these will be presented here.

CHRONIC ENDOCARDIAL VALVULAR DISEASES

Mitral Regurgitation.—In mitral regurgitation the ultimate brunt of the deformity falls to the right ventricle, which hypertrophies. Electrocardiograms are very apt to show preponderance of the right ventricle (Fig. 126).

Mitral Stenosis.—Electrocardiograms of patients with mitral stenosis are fairly uniform. In this lesion the left auricle and then the right ventricle bear the brunt of the burden, and, therefore, hypertrophy of these chambers occurs quite constantly. Owing to hypertrophy of the left auricle, the P wave is often increased in amplitude and frequently notched because of slight auricular asynchronism. These changes usually noted in Derivations

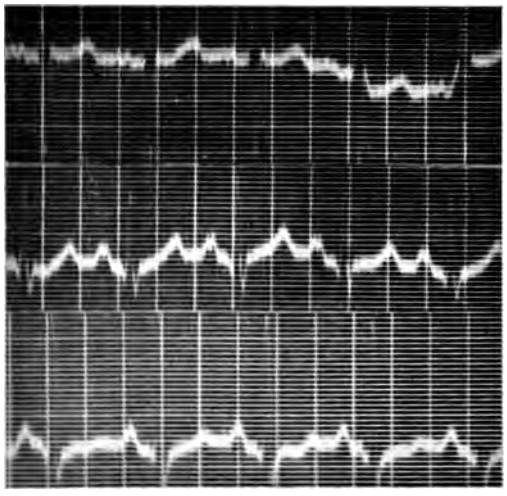

Fig. 126.—Electrocardiogram of patient with mitral regurgitation. Notched P wave in Derivation II. Right ventricular preponderance.

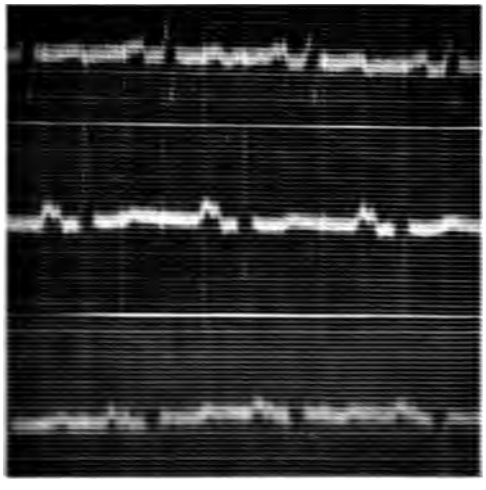

Fig. 127.—Electrocardiogram of patient with mitral stenosis. Notched P wave in Derivations I, II, and III.

II and III. Preponderance of the right ventricle is frequently present. Figs. 127, 128

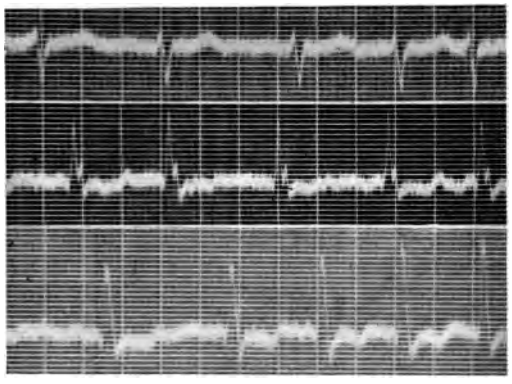

Fig. 128.—Electrocardiogram of patient with mitral stenosis. Auricular fibrillation; notching of Q R S complex in Derivations II and III; right ventricular preponderance.

Aortic Regurgitation.—In all aortic lesions the load falls to the left ventricle, which quite early undergoes hypertrophy. The electrocardiograms of aortic regurgitation quite constantly reveal

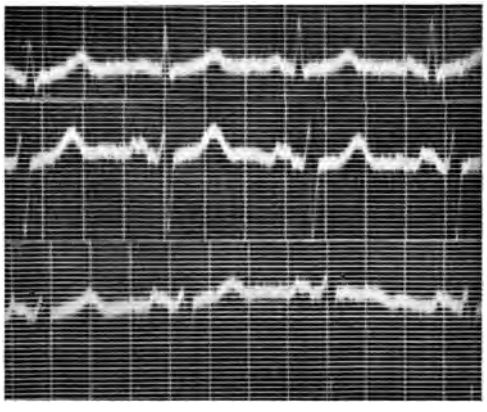

Fig. 129.—Electrocardiogram of patient with aortic regurgitation. Notched P wave in Derivation II. Marked left ventricular preponderance.

preponderance of the left ventricle. Angular definition of the individual waves is noted, and at times exaggeration of the T wave amplitude (Fig. 129).

Aortic Stenosis.—Preponderance of the left ventricle is the rule

with aortic stenosis, although at times the electrocardiograms reveal no imbalance. No characteristic graphic abnormalities occur (Fig. 130).

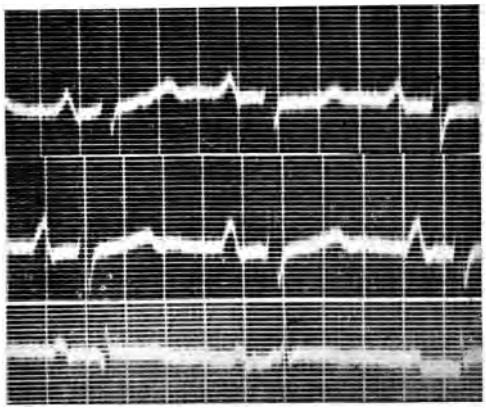

Fig. 130.—Electrocardiogram of patient with aortic stenosis. Exaggerated P wave in Derivations I and II. Notched P wave in Derivation III. Delayed auriculoventricular conduction, P–R interval 0.24 second.

Aortitis.—Preponderance of the left ventricle often occurs (Fig. 131).

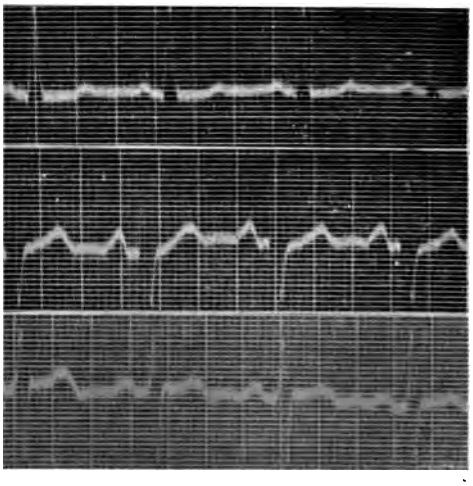

Fig. 131.—Electrocardiogram of patient with syphilitic aortitis. Left ventricular preponderance.

Aortic Aneurysm.—As in other aortic diseases, aneurysm frequently reveals preponderance of the left ventricle in the electrocardiogram. Figure 132 is an electrocardiogram of a patient with

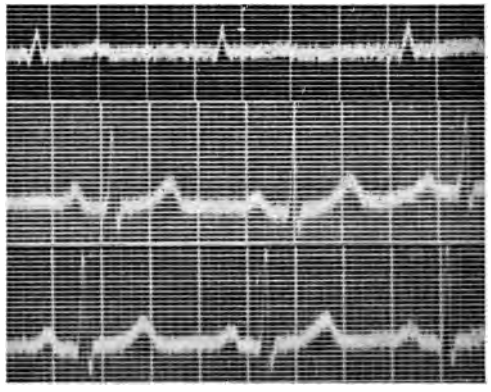

Fig. 132.—Electrocardiogram of a patient with an aneurysm of the aortic arch. Preponderance of the left ventricle.

an aneurysm of the aortic arch. The tracing is practically normal in all respects. Figure 133 is an electrocardiogram of a patient

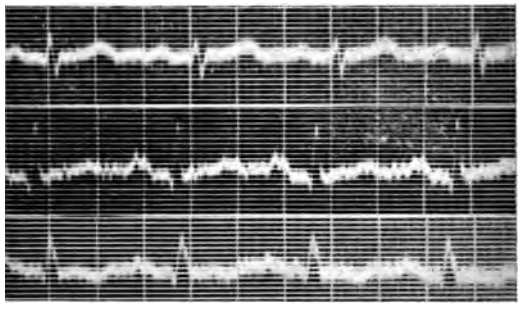

Fig. 133.—Electrocardiogram of patient with an aneurysm of the descending aorta. Slurring of the Q R S complex in Derivation I. Notching of the Q R S complex in Derivation III.

with an aneurysm of the descending aorta. The Q R S complexes in Derivation I are slurred, while those in Derivation III are definitely notched. The P wave in Derivations II and III is notched.

These abnormalities certainly are not characteristic. Figure 134 is an electrocardiogram of a patient with an aneurysm of the

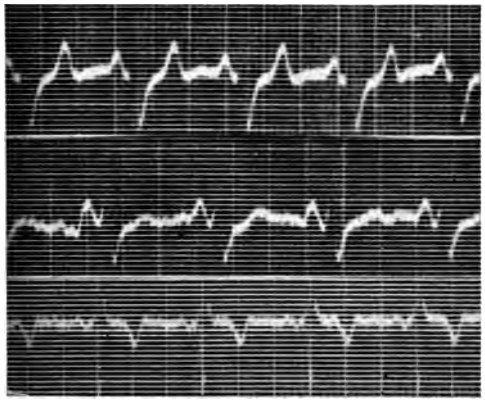

Fig. 134.—Electrocardiogram of patient with an aneurysm of the abdominal aorta. Exaggerated T wave in Derivation I. Exaggerated P wave in Derivation II. Negative T wave in Derivation III. Left ventricular preponderance.

abdominal aorta. The amplitude of the positive T wave in Derivation I and of the negative T wave in Derivation III is increased.

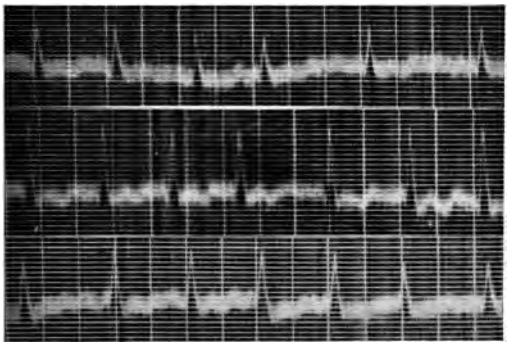

Fig. 135.—Electrocardiogram of patient with adhesive pericarditis. Auricular fibrillation. Negative T wave in Derivations II and III.

The P wave in Derivation II is exaggerated. There is preponderance of the left ventricle.

Chronic Adhesive Pericarditis.—Chronic adhesive pericarditis is usually associated with varying degrees of myocarditis and, therefore, the electrocardiograms of this disease vary greatly. Figure 135 is the electrocardiogram of a patient with chronic adhesive pericarditis who died soon after the tracing was taken. Auricular fibrillation is present, associated with T wave negativity in combined Derivations II and III.

Pericarditis with Effusion.—The effect of a pericardial effusion on the myocardium is dependent on several factors, such as the

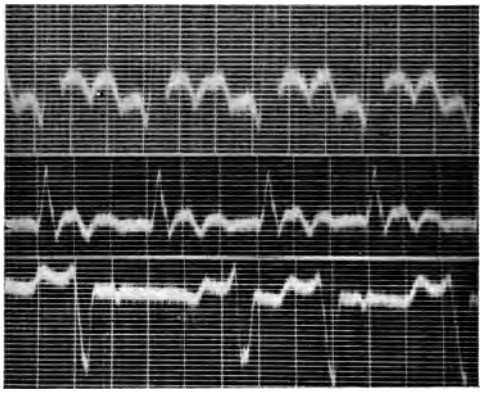

Fig. 136.—Electrocardiogram of patient with pericarditis with effusion. Occasional nodal premature contractions. Aberrant Q R S complexes in all derivations. Negative T wave in Derivation I. Left ventricular preponderance.

suddenness of fluid accumulation, and the degree of involvement of the myocardium by the infection responsible for the pericarditis.

The effect of a rapidly developing and progressive effusion is obvious. The cardiac mechanism does not have sufficient time to adjust itself to the encumberance, and by mechanical interference contraction amplitude is diminished and rate accelerates progressively in the attempt to maintain volume flow. An electrocardiogram of such a case would reveal a progressive tachycardia with deflections of low amplitude.

Chronic effusions or those of gradual accumulation rarely assume the proportions of those of sudden onset, and the heart may be embarrassed but little. This is due to the fact that less pressure is exerted by the effusion, and because of its gradual onset the heart is able to adjust its mechanism to the abnormal status. Electrocardiograms vary with the degree of myocardial damage or fatigue that eixsts. Figure 136 is the electrocardiogram of a patient with chronic pericarditis with effusion. Note the aberrant Q R S complexes in all derivations. The T wave in Derivation I is negative. Preponderance of the left ventricle is present.

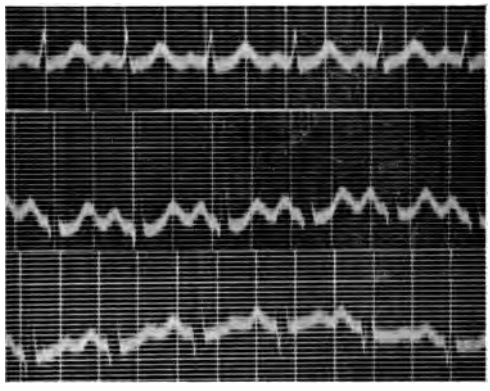

Fig. 137.—Electrocardiogram of a patient with early exophthalmic goiter. Note the rolling contour of the cycles and the increased amplitude of deflections, especially of the T wave.

Hyperthyroidism.—The effect of hyperthyroidism on the heart is twofold: (1) the effect of the thyroid active principle, thyroxin, on the myocardium, which is cellular, and (2) the increased cardiac work accompanying the elevation of the basal metabolic rate.

Electrocardiograms of patients with exophthalmic goiter (Fig. 137) or with adenoma with hyperthyroidism are modified largely by the degree of myocardial degeneration. Auricular fibrillation is a very frequent phenomenon, often disappearing after thyroidectomy when the basal metabolic rate attains normal.

Myxedema.—Electrocardiograms of patients with myxedema usually reveal deflections of low amplitude, particularly of the T wave. Figure 138 is the electrocardiogram of a patient suffering from high-grade myxedema. Note the low amplitude deflections. The T wave in Derivation III is negative.

Angina Pectoris.—Angina pectoris does not produce characteristic electrocardiograms, although group study reveals certain abnormalities which occur quite constantly. Data in a series of 155 cases of angina pectoris recently published showed that coronary

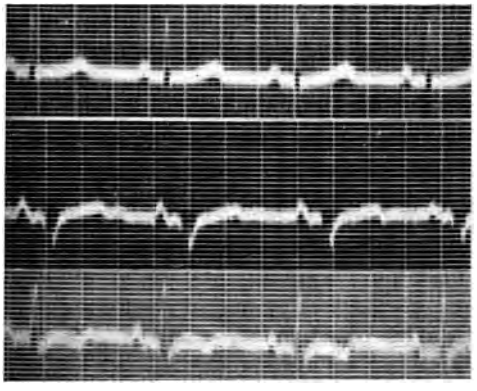

Fig. 138.—Electrocardiogram of a patient with high-grade myxedema.

disease occurred in 86.5 per cent. of cases, aortic disease in 12.2 per cent., and mitral stenosis in 1.3 per cent.

Thirty electrocardiograms (19.4 per cent.), including ventricular preponderance and T wave negativity in Derivation III, were considered normal.

Changes in the Final Ventricular T Wave.—Abrupt peaked positive T waves of exaggerated amplitude occurred in 11.6 per cent. of the electrocardiograms. T wave negativity occurring in isolated and combined derivations was present in 51.6 per cent. of the electrocardiograms. No significance can be attached to

T wave negativity in Derivation III alone, as it occurs in normal and in diseased hearts. Excluding T wave negativity in Derivation III, 51 patients (32.9 per cent.) had significant T wave negativity. T wave negativity in Derivation I occurred in 25 (16.1 per cent.) electrocardiograms, in combined Derivations II and III in 12 (8.8 per cent.), in combined Derivations I and II in 8 (5.2 per cent.), and in combined Derivations I, II, and III in 5 (3.2 per cent.). The significance of these four types of T wave negativity is appreciated when the mortality statistics are analyzed. Information has been received concerning 37 of these patients; 26 (70.3 per cent.) have died from heart disease during a period of five years; 6 are known to have died in anginal attacks. The mortality was 72 per cent. in Derivation I, 66.6 per cent. in combined Derivations II and III, 75 per cent. in combined Derivations I and II, and 80.8 per cent. in combined Derivations I, II, and III.

Aberrant Q R S Complexes in All Derivations.—In this series 22 (14.2 per cent.) patients had aberrant Q R S complexes in all derivations of their electrocardiograms; 16 of these records were associated with significant T wave negativity. T wave negativity in Derivation I occurred with greatest frequency, being present in 8 (36.4 per cent.) of these electrocardiograms.

A high mortality occurs likewise in this group. Information has been received regarding 16 patients; 10 (62.5 per cent.) have died from heart disease, and 4 of these died in anginal attacks.

Aberrant Q R S Complexes in Isolated Derivations.—Notching or slurring of the Q R S complex in isolated derivations occurred in 59 (37 per cent.) tracings; 33 (21.2 per cent.) patients had notched Q R S complexes in isolated derivations of their electrocardiograms. The greatest number, 19 (57.5 per cent.), occurred in Derivation III. In order of frequency the other derivations were involved as follows: Derivation II, 9 (27.9 per cent.), combined Derivations II and III, 4 (12.1 per cent.), and Derivation I,

1 (3 per cent.); 5 (15.1 per cent.) of these electrocardiograms were associated with significant T wave negativity. Excluding these cases the cardiac mortality in the group was 47.3 per cent.

Twenty-six (16.7 per cent.) patients had slurred Q R S complexes. In 10 (38.4 per cent.) patients these findings occurred in Derivation II, in 8 (30.7 per cent.) in Derivation I, in 7 (26.9 per cent.) in Derivation III, and in 1 patient (3.8 per cent.) in combined Derivations I and II. Eleven (42.3 per cent.) of these electrocardiograms were associated with significant T wave negativity. Excluding the cases with significant T wave negativity the cardiac mortality was 20 per cent.

Delayed Auriculoventricular Conduction.—Prolongation of the P–R interval beyond 0.22 second is abnormal and indicates delay in impulse transmission between auricles and ventricles. Only 2 patients (1.3 per cent.) had this abnormality in their electrocardiograms. In both cases the P–R interval was 0.24 second. In one electrocardiogram negativity of the T wave in Derivation I occurred. Information was obtained regarding one of these patients, who died in an anginal attack four years after examination.

Complete Auriculoventricular Dissociation.—Only one patient (0.6 per cent.) had complete auriculoventricular dissociation. The auricular rate was 94 and the ventricular rate 47 each minute. This patient died of heart disease seven and one-half months after examination.

Auricular Fibrillation.—Three patients (1.9 per cent.) had auricular fibrillation. The infrequency of auricular fibrillation with angina pectoris is apparent and due undoubtedly to the fact that the ventricles bear the brunt of the ravages of the disease. Information has been received regarding 2 of these patients; both have died from heart disease, one two weeks and the other one year after examination.

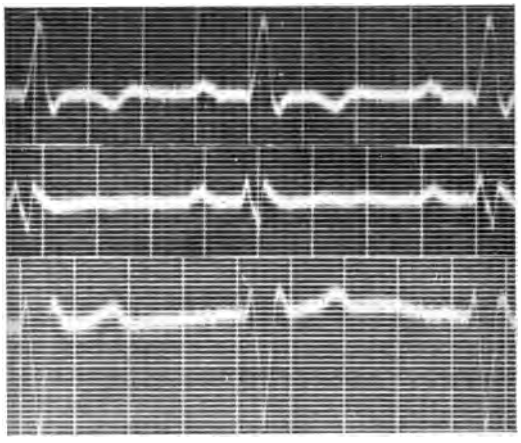

Fig. 139.—Patient had had angina pectoris for two years. Died in anginal attack two weeks after examination.

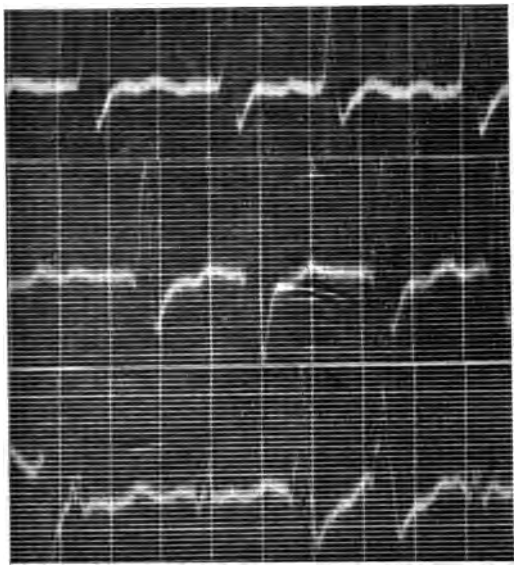

Fig. 140.—Patient had had angina pectoris for six months. Died of heart disease two weeks after examination.

Cardiac Mortality.—The cardiac mortality of the complete group was 46.7 per cent. It is interesting to contrast this per-

centage with the mortality attending significant T wave negativity (70.3 per cent.) and aberrant Q R S complexes in all derivations

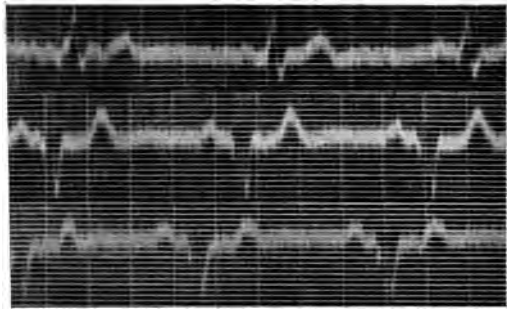

Fig. 141.—Patient had had angina pectoris for one year. Died in anginal attack two weeks after examination.

(62.5 per cent.); 16 patients are known to have died in anginal attacks; the information regarding the others was not specific.

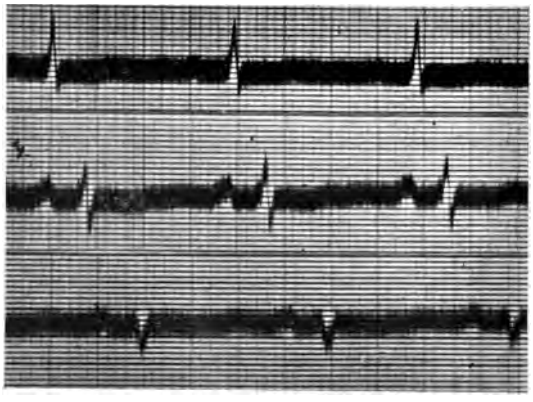

Fig. 142.—Patient had had angina pectoris for six years. Died in anginal attack two months after examination.

There was no constant relationship between the duration of angina and the degree of electrocardiographic abnormality. Figures 139 to 185 are the electrocardiograms of the fatal cases.

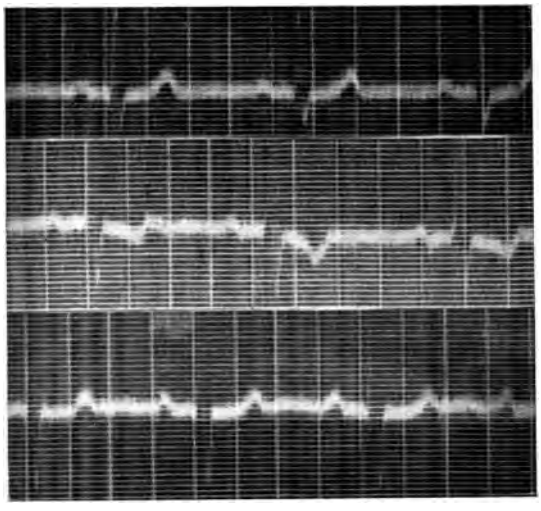

Fig. 143.—Patient had had angina pectoris for one and one-half years. Died in anginal attack two and one-half months after examination.

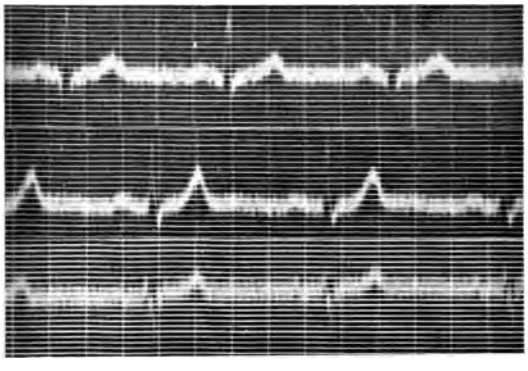

Fig. 144.—Patient had had angina pectoris for two months. Died of heart disease two and one-half months after examination.

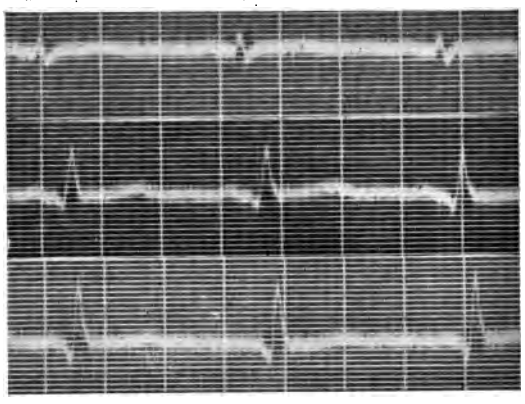

Fig. 145.—Patient had had angina pectoris for nine months. Died three months after examination (aortic aneurysm).

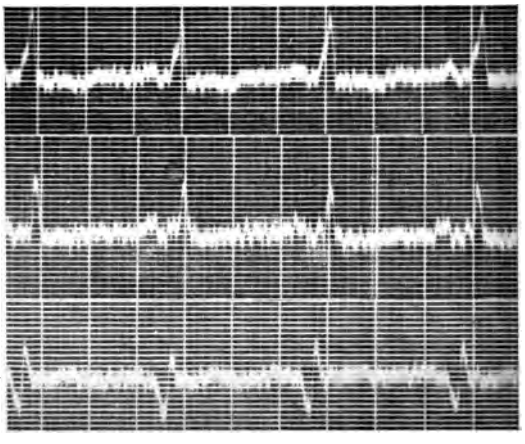

Fig. 146.—Patient had had angina pectoris for two years. Died in anginal attack three months after examination.

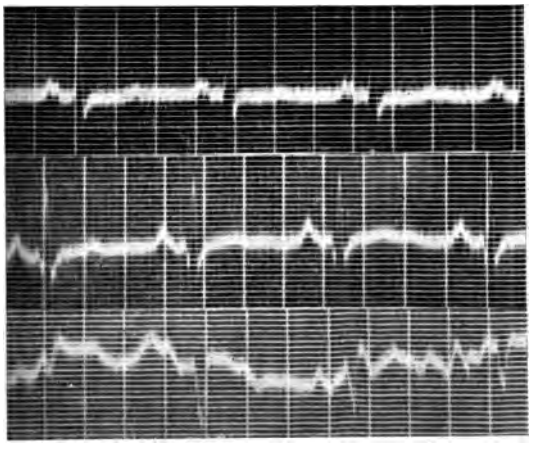

Fig. 147.—Patient had had angina pectoris for two and one-half months. Died in anginal attack three months after examination.

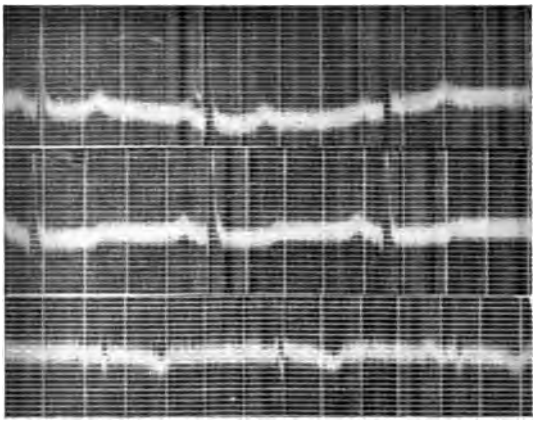

Fig. 148.—Patient had had angina pectoris for seven months. Died in anginal attack three months after examination.

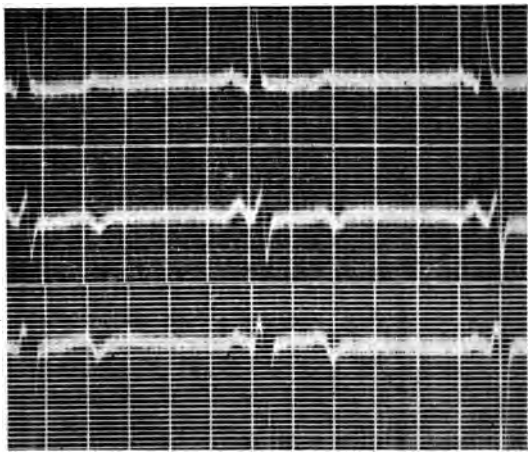

Fig. 149.—The duration of angina pectoris unknown. The patient died of heart disease three and one-half months after examination.

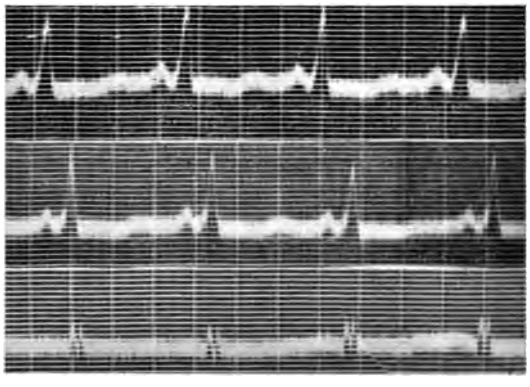

Fig. 150.—Patient had had angina pectoris for four years. Died of heart disease four months after examination.

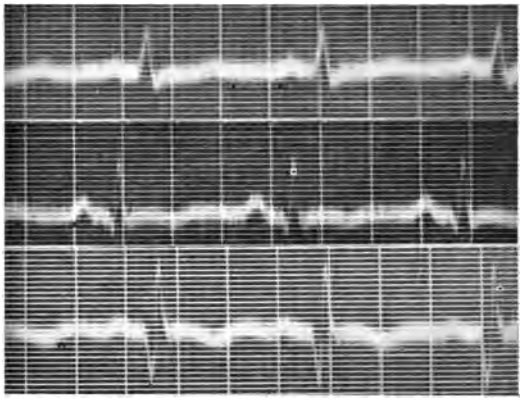

Fig. 151.—Patient had had angina pectoris two and one-half years. Died in anginal attack four months after examination.

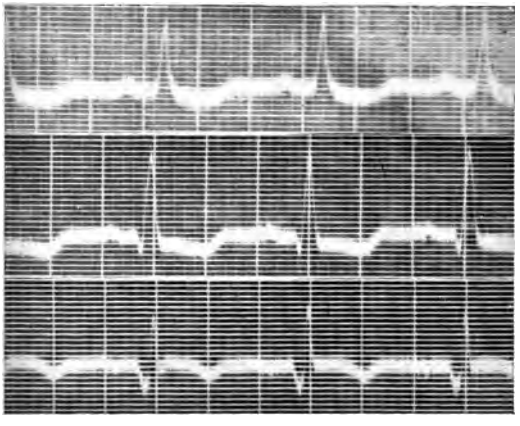

Fig. 152.—Patient had had angina pectoris for three years. Died of heart disease four and one-half months after examination.

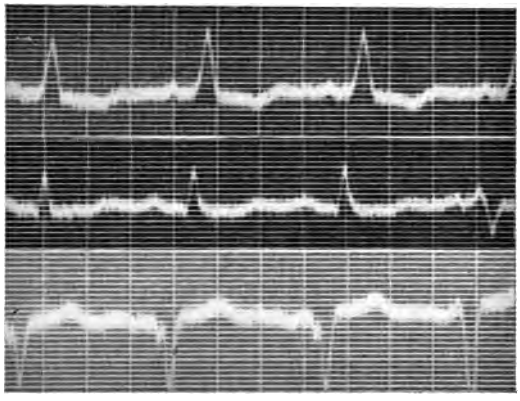

Fig. 153.—Patient had had angina pectoris for eight years. Died of heart disease five months after examination.

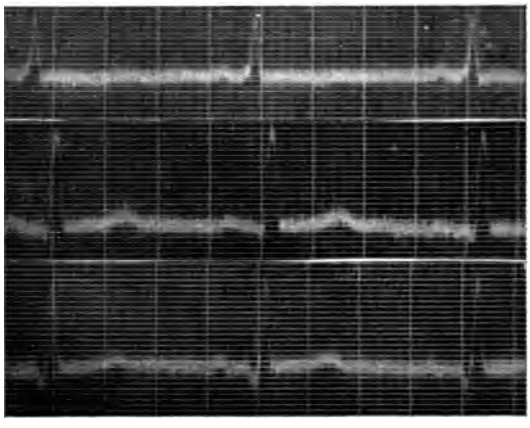

Fig. 154.—Patient had had angina pectoris for two years. Died in anginal attack five months after examination.

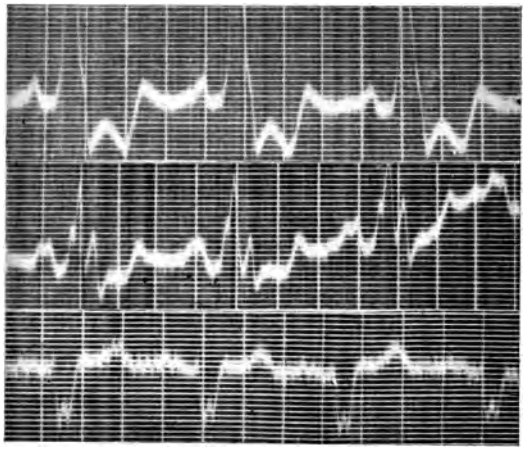

Fig. 155.—Patient had had angina pectoris for seven months. Died of heart disease six months after examination.

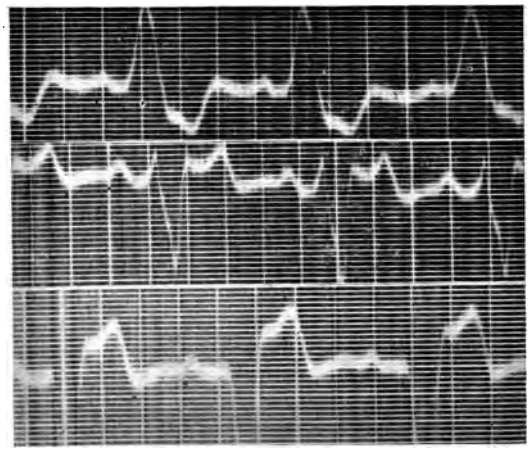

Fig. 156.—The duration of angina pectoris not known. The patient died of heart disease six and one-half months after examination.

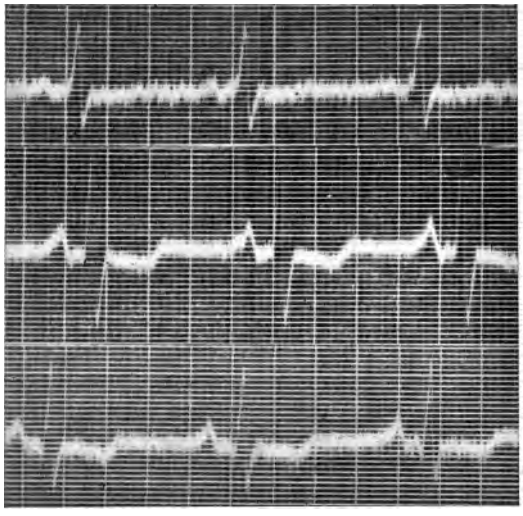

Fig. 157.—Patient had had angina pectoris for five months. Died of heart disease seven months after examination.

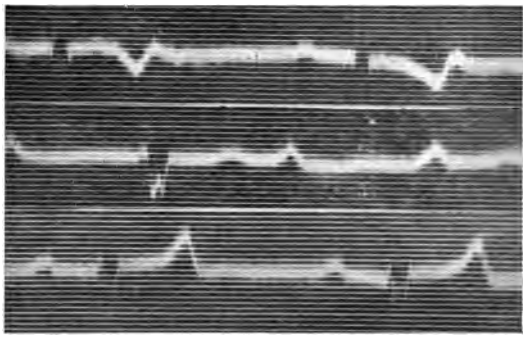

Fig. 158.—Patient had had angina pectoris for three years. Died of heart disease seven and one-half months after examination.

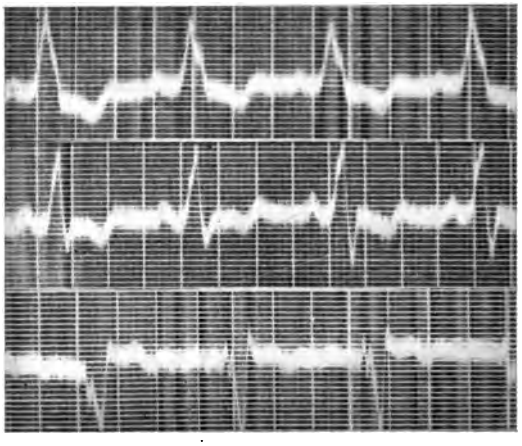

Fig. 159.—Patient had had angina pectoris for ten months. Died in anginal attack nine months after examination.

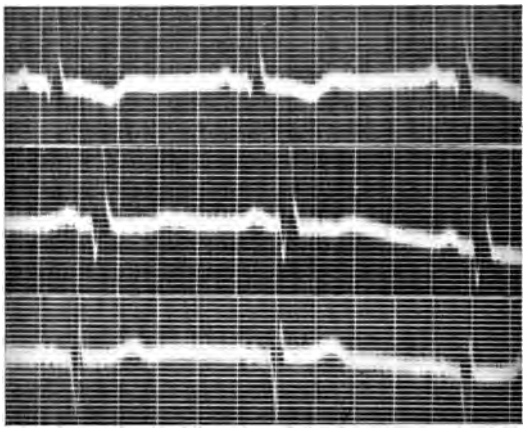

Fig. 160.—Patient had had angina pectoris for five years. Died of heart disease eleven months after examination.

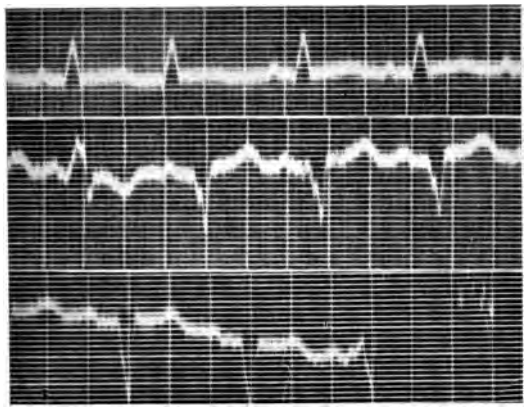

Fig. 161.—Patient had had angina pectoris for five months. Died in anginal attack eleven months after examination.

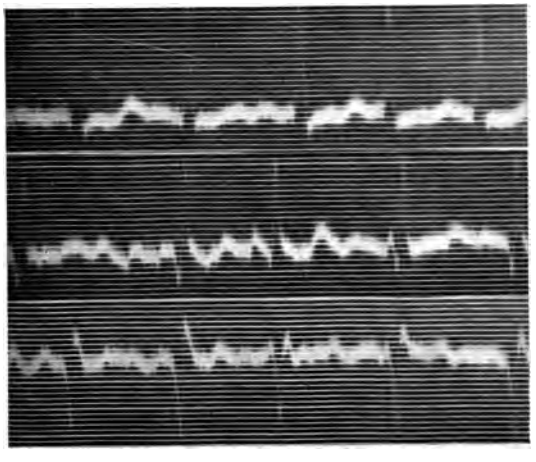

Fig. 162.—Patient had had angina pectoris for three weeks. Died of heart disease one year after examination.

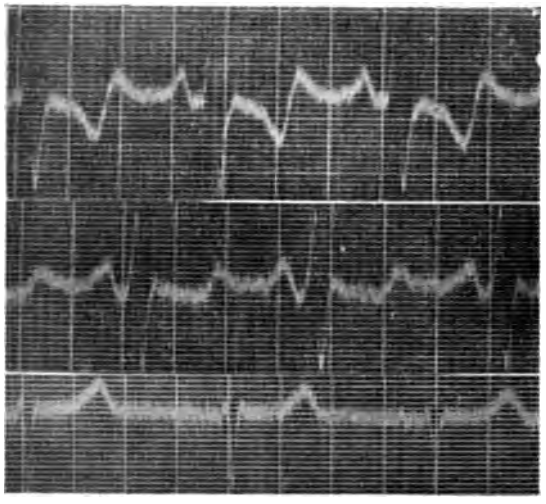

Fig. 163.—Patient had had angina pectoris for one year. Died of heart disease one year after examination.

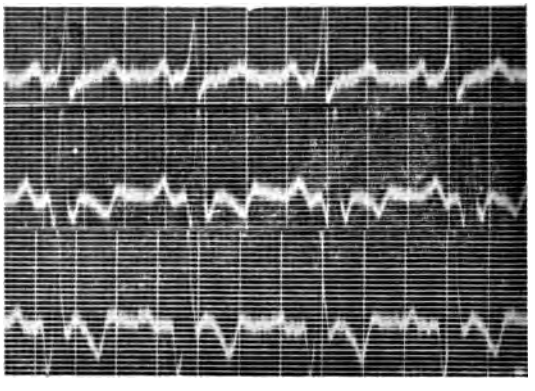

Fig. 164.—Patient had had angina pectoris for three weeks. Died of heart disease thirteen months after examination.

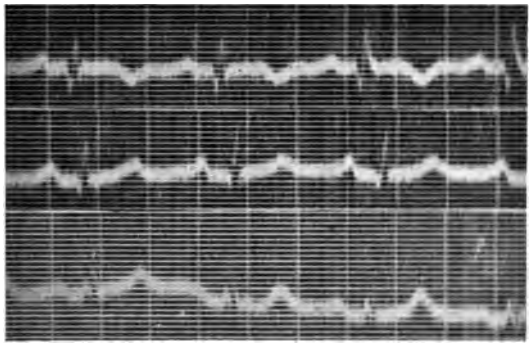

Fig. 165.—Patient had had angina pectoris for six weeks. Died of heart disease fourteen months after examination.

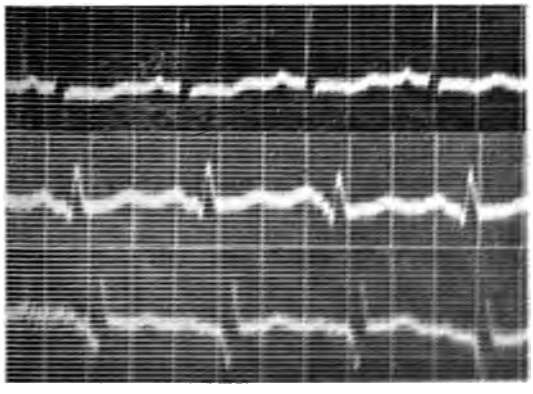

Fig. 166.—Patient had had angina pectoris for two years. Died in anginal attack fifteen months after examination.

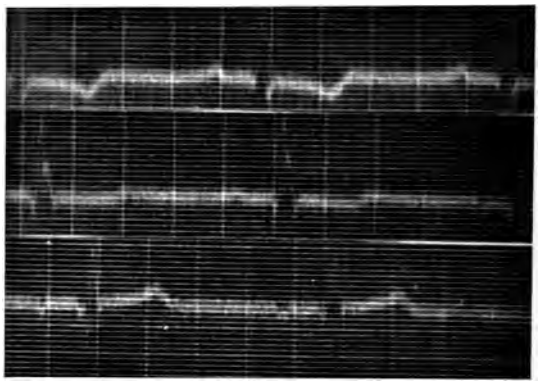

Fig. 167.—Patient had had angina pectoris for five years. Died of heart disease sixteen months after examination.

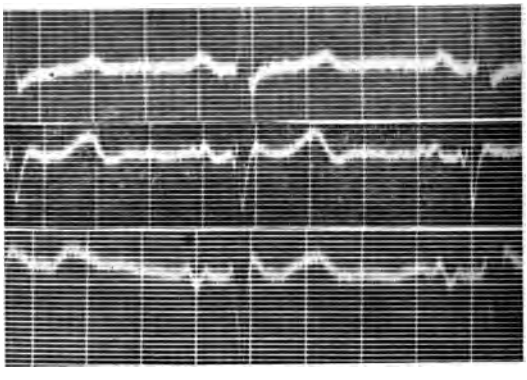

Fig. 168.—Patient had had angina pectoris for five years. Died of heart disease two years and four months after examination.

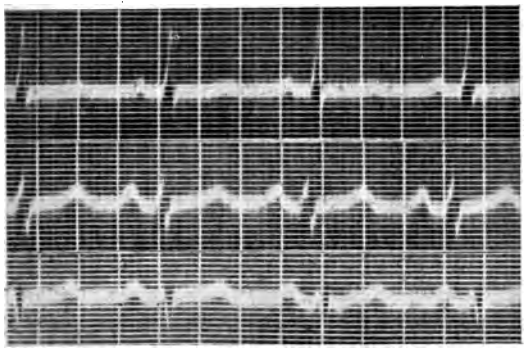

Fig. 169.—Patient had had angina pectoris for eight months. Died in anginal attack twenty months after examination.

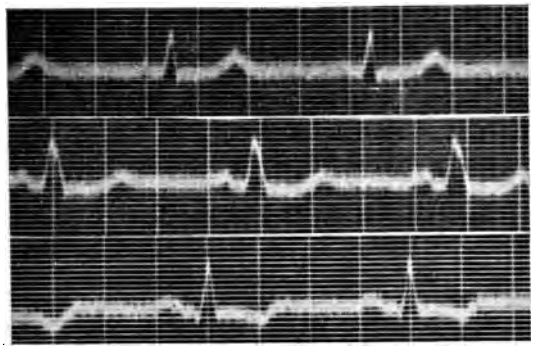

Fig. 170.—Patient had had angina pectoris for twenty years. Died of heart disease twenty months after examination.

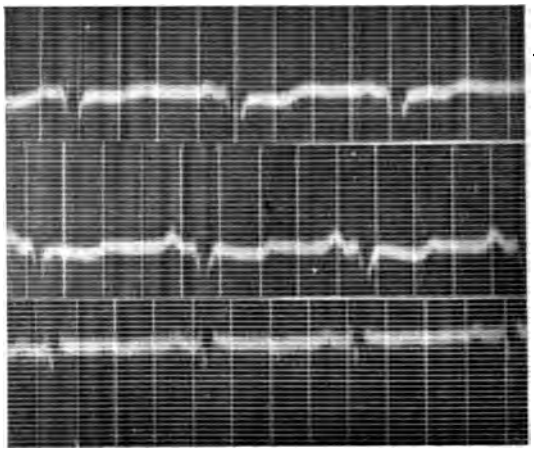

Fig. 171.—Patient had had angina pectoris for five years. Died of heart disease twenty months after examination.

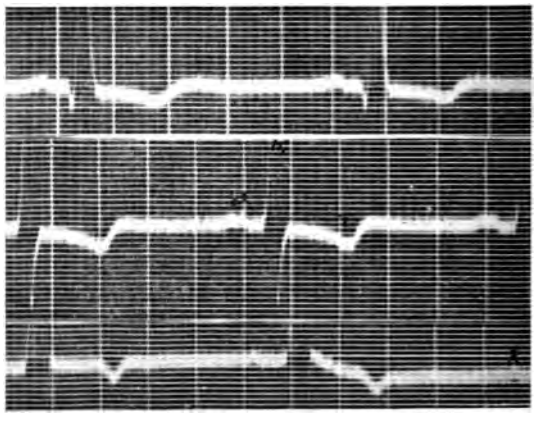

Fig. 172.—Patient had had angina pectoris for four years. Died in anginal attack twenty-one months after examination.

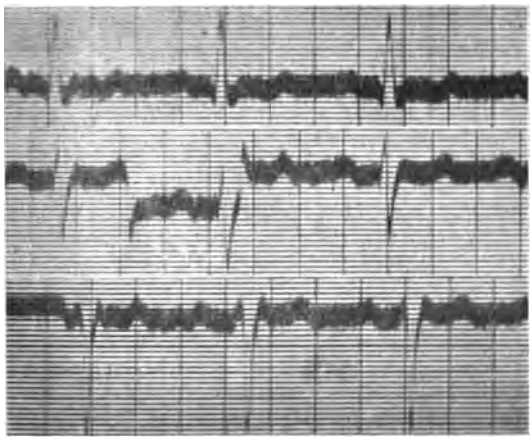

Fig 173.—Patient had had angina pectoris for five years. Died of heart disease two years after examination.

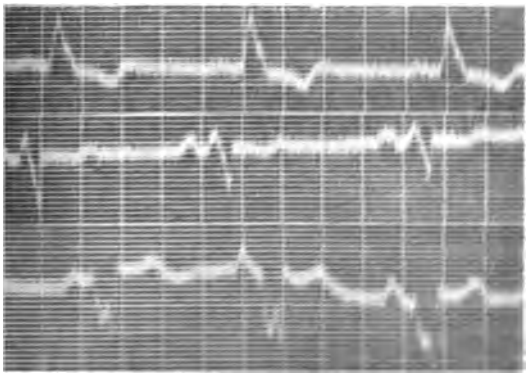

Fig. 174.—Patient had had angina pectoris one and one half years. Died of heart disease two years after examination.

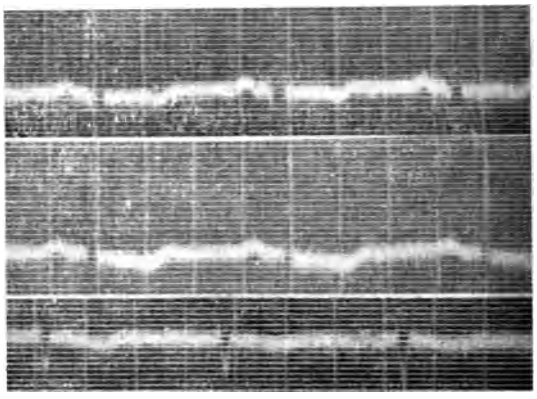

Fig. 175.—Patient had had angina pectoris for one year. Died of heart disease three years after examination.

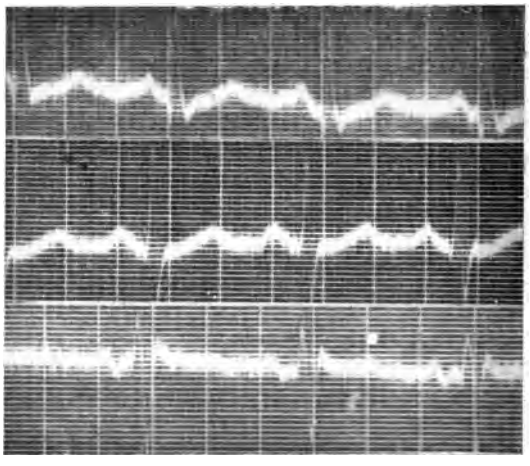

Fig. 176.—The duration of angina pectoris not known. The patient died of heart disease three years and two months after examination.

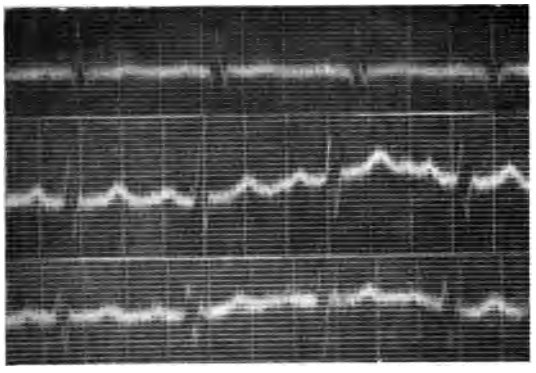

Fig. 177.—Patient had had angina pectoris for two years. Died in anginal attack three and one-half years after examination.

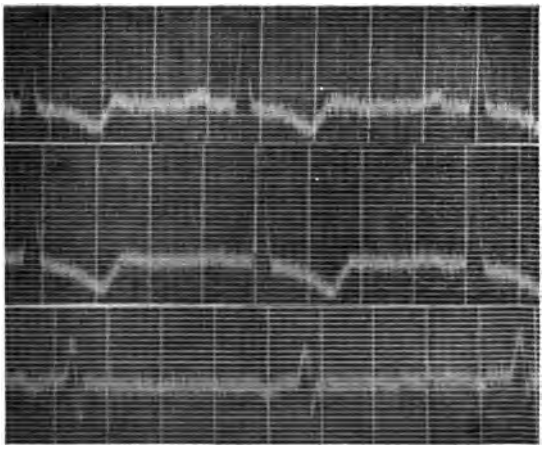

Fig. 178.—Patient had had angina pectoris for ten years. Died of heart disease three years and eight months after examination.

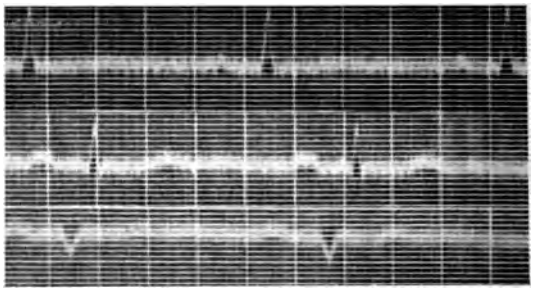

Fig. 179.—Patient had had angina pectoris for three years. Died in anginal attack four years after examination.

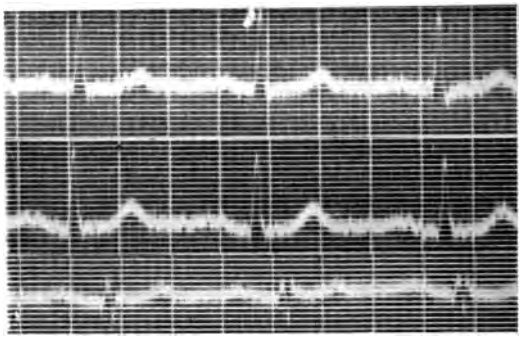

Fig. 180.—Patient had had angina pectoris for six weeks. Died of heart disease four and one-half years after examination.

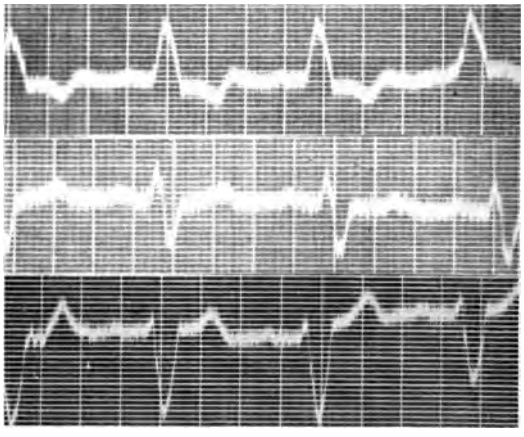

Fig. 181.—Patient had had angina pectoris for two years. Died of heart disease. Date not known.

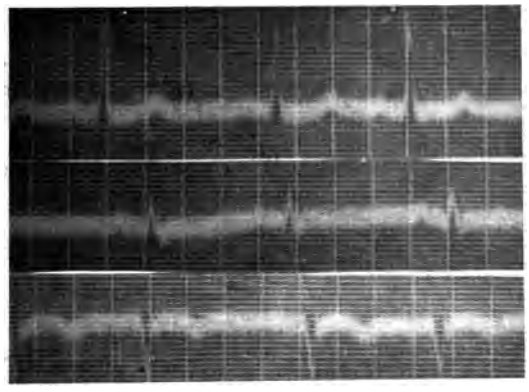

Fig. 182.—Patient had had angina pectoris for one year. Died of heart disease Date not known.

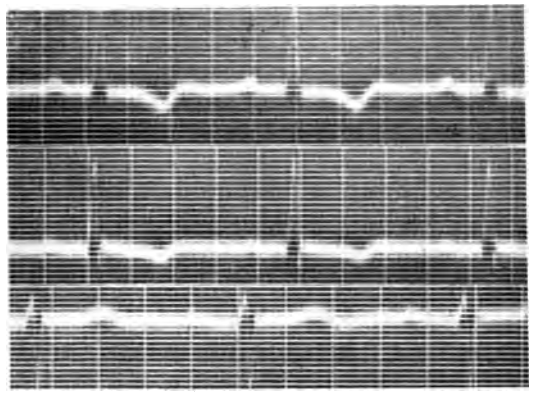

Fig. 183.—Patient had had angina pectoris for six months. Died of heart disease. Date not known.

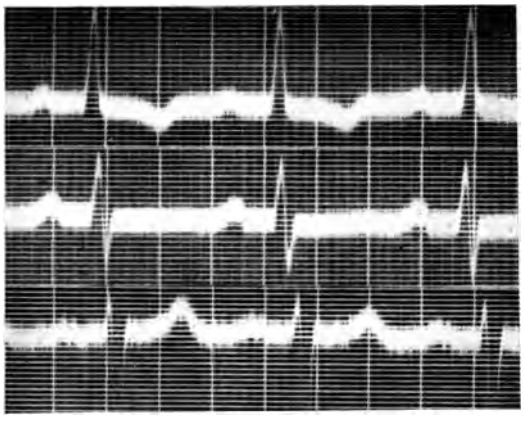

Fig. 184.—Patient had had angina pectoris for six months. Died of heart disease Date not known.

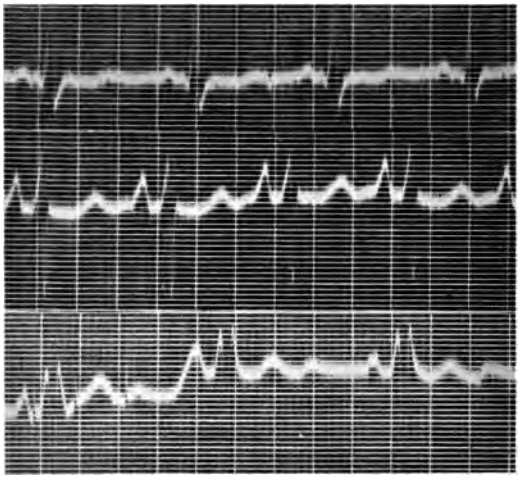

Fig. 185.—Patient had had angina pectoris for four years. Died of heart disease. Date not known.

Bibliography

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